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BEFORE THE ANNUAL 1945 MEETING
of the
AMERICAN SURGICAL ASSOCIATION

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ANNALS OF SURGERY

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A request to Director Byrnes that the American Surgical Association be allowed to hold its meeting in May was politely but firmly refused. The Council has decided that the papers prepared for this meeting be published as a unit, without discussion, as proceedings of the Association.

William Darrach
President

THE PROBLEM OF PORTAL HYPERTENSION IN RELATION TO THE HEPATOSPLENOPATHIES*

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IT MAY BE PERTINENT, in introducing the topic of this discussion, to say a few words about our interest and experience in the study of portal hypertension. Some 17 years ago, when the College of Physicians and Surgeons of Columbia University and the Presbyterian Hospital moved to their new common site, a group of physicians, surgeons and pathologists organized one of the several combined clinics. This soon acquired the name of the Spleen Clinic. Its purpose is to study patients with splenopathies before, during and after whatever treatment is decided upon by agreement of the group. The essential feature of this combined clinic is the laboratory, where all the blood studies and other tests are done by the same expert technicians on the patients, whether as out-patients, in-patients, or in follow-up visits. These patients are referred to the Spleen Clinic from the Vanderbilt Clinic, the Out-patient Department of the Columbia-Presbyterian Medical Center, and by other physicians; and after study and treatment they are returned to the referring physician with the request to have the patients return to the Spleen Clinic for indefinite follow-up studies[1] (Fig. 1).

As a result, there have been studied not only patients with splenopathies but many with disorders of the hematopoietic system and combined diseases of the liver and spleen. Table I is a summary of the splenopathies and the hepatosplenopathies that have been studied and followed in this Clinic to March 15, 1945.

No one can work for any length of time in a clinic of this kind, with

* The E. Starr Judd Lecture. Read at The Medical School, University of Minnesota, April 10, 1945.

. This paper was to have been presented before the Annual Meeting of the American Surgical Association, May, 1945.

physicians, surgeons and pathologists having a common purpose, without acquiring valuable experience and developing an interest in the diagnosis, treatment and end-results of these lesions. A common language is spoken and there are no miracles among friends. Again, I would emphasize the importance of having the accurate hematologic and other laboratory studies done by the same group of experts throughout the study of these patients, before, during and after treatment.

THE PORTAL SYSTEM

There are many marked differences between the portal and systemic venous systems, both anatomically and physiologically. The portal venous

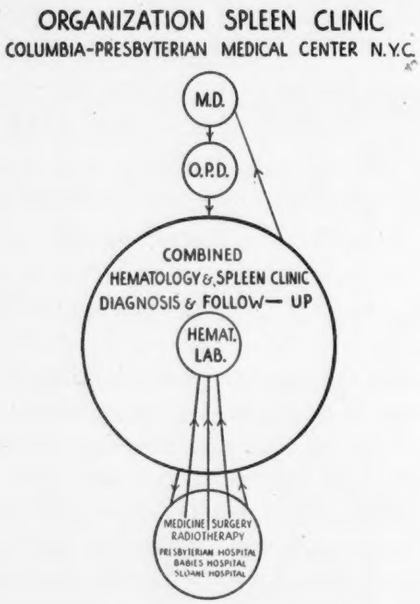


FIG. 1

system is interposed between two capillary beds. It drains the capillaries of the gastro-intestinal tract, the pancreas and the gallbladder, and the venous sinuses of the spleen; and empties into the capillary network, or sinusoids, of the liver. It has no valves. The portal vein carries about 75 per cent of the blood emptying into the liver. In this blood it carries nutrient material from the gastro-intestinal tract, and insulin from the pancreas, to be altered, detoxified and utilized by the liver, but it carries very little oxygen. For this the liver is dependent upon the hepatic artery which conveys the remaining 25 per cent of the blood entering the liver.

To understand the pathogenesis, the pathology, the diagnosis and the treatment of portal hypertension it is essential to review certain points in the anatomy and physiology of the circulation of the liver and the spleen. Unfortunately, there is still considerable controversy regarding some of these points; an endeavor is made to present as much of the new and accepted data as possible.

THE LIVER LOBULE IN RELATION TO THE PORTAL AND HEPATIC CIRCULATION

The mammalian liver is made up of small polygonal masses, each of which represents the architectural unit, or lobule, 0.7 to 2 mm. in diameter. In some mammals, as the pig, each lobule is completely surrounded by a layer of connective tissue, but in man these connective tissue partitions are poorly developed, resulting in less well-defined lobules. Furthermore, the liver lobule is determined by the arrangement of the blood vessels rather than by the duct system. The lobule is a polygonal prism, with five, six or seven sides, with the vertical diameter several times greater than the transverse. Running through the center of the lobule, in its long axis, is the central vein, while at the periphery are the branches of the portal vein with a lymphatic network, the branches of the hepatic artery and the interlobular bile ducts. The latter structures are enclosed in the connective tissue of Glisson's capsule.

Separating the central vein and the vessels in Glisson's capsule are the hepatic cells, arranged in cords with the sinusoids or capillaries running between the cords of cells and connecting the portal vein radicals and hepatic arterial terminals with the central vein. These central veins unite as intercalated veins to empty into collecting veins which, in turn, end in the hepatic veins to join the vena cava (Fig. 2).

THE RELATIONS OF THE BLOOD FLOW OF THE HEPATIC ARTERY AND THE PORTAL VEIN

One of the earliest and most significant studies on these two circulations was made by Gad[2] in 1873, who wrote a dissertation based on experimental work, in which he concluded that the arterial circulation in the liver performed the dual function of bringing oxygenated blood to the liver and of mechanically controlling the portal flow. He also suggested an aspirating action of the arterial on the portal current as the former passed by the openings of the latter, joining with them at an acute angle. He demonstrated that a current through the arteries with increased pressure retarded the portal flow.

In 1907, Herrick[3] published his studies on the causes of increased portal pressure in portal cirrhosis. He perfused normal livers and cirrhotic livers through the hepatic artery and the portal vein, noting the effect of the volume flow, one on the other, at increasing pressures. He pointed out that in the normal liver there is:

1. In the portal vein a large volume circulation with a low pressure.
2. In the hepatic artery a small volume circulation with a high pressure.
3. A common channel of exit, the hepatic vein, for fluid entering by these two circulations.
4. A freely expansible tissue framework.
5. Two methods by which the entering circulations may influence each other, *i.e.,* by direct communication or by lateral pressure.
6. A vasomotor mechanism to both sets of vessels.

7. An equalization of these pressures at the junction of the interlobular and intralobular venules of the liver lobules which takes place through the communications between the veins coming from the capillaries, bringing arterial blood and the portal venules.

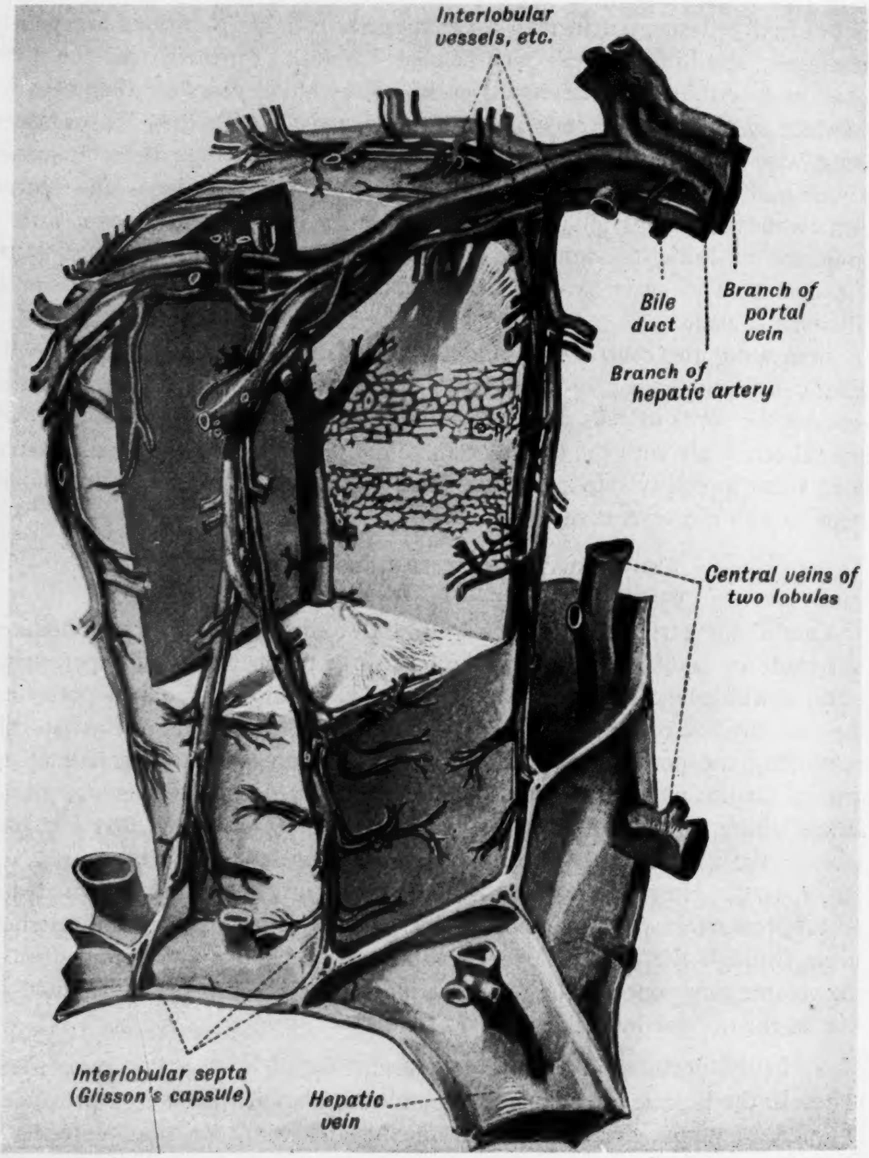


FIG. 2.—Lobule of the liver of a pig. Wax reconstruction by Vierling. A portion of the lobule is cut away to show the bile capillaries and sinusoids. ×400. After Braus. (From Maximow's "Textbook of Histology," 4th edition. Philadelphia, W. B. Saunders Co.)

In the normal liver Herrick found that the rise in portal pressure was 1 mm. for every 40 mm. of arterial pressure; whereas in the cirrhotic liver it was 1 mm. for every 6 mm. of arterial pressure. It was found that arterial

rise in pressure did not affect the portal pressure in normal livers until it had passed the 100 mm. mark. In cirrhotic livers the effect on the portal pressure was evident at 30 mm. of arterial pressure.

McIndoe,[15] in his studies of the vascular bed of cirrhotic livers, found that a large part of the fluid perfused through the portal vein escaped through the collaterals, and that 13 per cent was the largest amount recovered from the hepatic vein, as compared to 100 per cent in the normal liver. On the other hand, he found that a much greater proportion of fluid, perfused through the hepatic artery, in a cirrhotic liver, passes out through the hepatic vein. He was unable to duplicate Herrick's observations that the high pressure in the hepatic artery was responsible for increasing the portal pressure.

In advanced cases of portal cirrhosis with the portal blood passing through established collaterals the shunt is able to carry a certain load, but with increased portal pressure rupture of the collaterals may take place. In such cases the hepatic artery is conveying most of the portal blood to the parenchyma of the liver.

As the combination of increasing interlobular fibrosis with degeneration and regeneration of liver cells slowly progresses, there is a gradual shutting off of portal blood from the hepatic cells. When the fibrosis increases to the point of shutting off the arterial supply parenchymal insufficiency develops. McIndoe points out that the duration of the period from the time of complete diversion of portal blood into collateral channels to the obliteration of the persisting arterial supply to the remaining liver cells is longer or shorter according to the rate of sclerosis of the intrahepatic vascular bed and the varying degree of involvement of the two circuits. This explains the variable periods of hemorrhage and of survival in cirrhotic patients.

McIndoe's analysis of the obstructive factor in cirrhotic livers questions the conclusions of Herrick in his perfusion experiments and throws new light on the rôle of the collateral channels in intrahepatic portal bed block. This is an important field for further investigation with new methods of study in the living animal with experimentally produced cirrhosis.

THE VASCULAR BED OF THE SPLEEN

The following discussion is abstracted from studies made in our laboratory[4, 5]:

Following Malpighi's[6] classic description of the anatomy of the mammalian spleen, in 1666, there has been a recurrent controversy among anatomists regarding the histology of the vascular bed of that organ. Proponents of an open circulation claimed that there was no continuity between the terminal arterioles and the venous sinuses, but that the splenic pulp spaces provide the only link between the arterial and venous radicals. Equally emphatic in their opposing claims have been those who maintained that the arterioles emptied directly into the venous sinuses and that the vascular bed was a closed system. Modern opinion rather favors a combined type of circulation

in the mammalian spleen, that is, an open component in the vascular bed which permits the flooding of the splenic pulp spaces with whole blood but with additional pathways available, by means of which the extravascular detour of the pulp spaces may be short-circuited by direct communications between the arterioles and the venous sinuses. Whether these arteriovenous connections of which it is composed are structurally intact vascular tubes or whether their appearance may be accounted for on a purely functional basis, such as an alteration in the porosity of the pulp caused by contraction of the splenic musculature, is considered to be an open question.

The reasons for this three-sided debate have been the difficulty of interpreting the intricate patterns of the spleen as seen in microscopic sections stained by different methods, and the varying results obtained by the several technics of injecting the vascular bed of the spleen both by artery and by vein.

In 1931, McNee[7] reported that "in the direct observations of the thin edge of a mouse's spleen, examined with a dissecting microscope and very powerful light, erythrocytes could be seen lying stationary in the pulp while blood was pumping freely through the adjacent arterioles into sinuses and then emerging by the veins." No one made use of this valuable suggestion in method until Knisely,[8] in 1934 and 1936, working in the Hull Laboratory, in the University of Chicago, published his studies on transilluminated living spleens. He described the finest structural and functional details of the vascular bed; and his account of them was expressed in no uncertain terms. His conclusion that the "unstimulated splenic vascular system of mice, rats and cats consists of a system of preformed, interconnected lined channels," normally intact and permitting the egress of only the fluid content of the blood, presented the most convincing argument in favor of the hypothesis of the closed circulatory mechanism in the spleen. He maintained that trauma incidental to manipulation, injection and fixation of the spleen had been responsible for the antagonistic studies of histologists in this field.

Knisely's positive description of the visualized functioning circulation in the spleen and his graphic schematic drawings of the venous sinuses in what he termed the three phases of filling, concentrating and emptying of red blood cells, with sphincters at the entrance and exit of the sinuses, were so convincing that several writers on the subject of splenic circulation have recently accepted his claims without question. Von Herrath,[9] in his paper published in 1938, speaks of the "hematocrit function" of the venous sinuses as demonstrated by Knisely. But no one, until we began this study, had repeated Knisely's observations or corroborated or questioned his conclusions.

Because many of the problems in the pathology and clinical manifestations of the splenopathies depend upon a knowledge of the exact mechanism of the circulation in the spleen, the members of the Spleen Clinic at the Columbia-Presbyterian Medical Center were especially interested in Knisely's papers.[8] We visited the Hull Laboratory on two occasions and later sent one of our Surgical Residents, Dr. David W. MacKenzie, Jr., for a period of

four months to study with Dr. Knisely. He most kindly gave Dr. MacKenzie every opportunity, and on his return to our laboratory the apparatus and method were set-up for studying the transilluminated spleen.

Before studying abnormal spleens it was essential for us to become thoroughly familiar with the circulation in the normal spleen. It had become apparent to Doctor MacKenzie that two fundamental faults in Knisely's technic made a continuous uninterrupted study of vascular fields impossible: The first, and most misleading, was the constantly moving visual field caused by the respiratory movements transmitted to the spleen under the microscope;

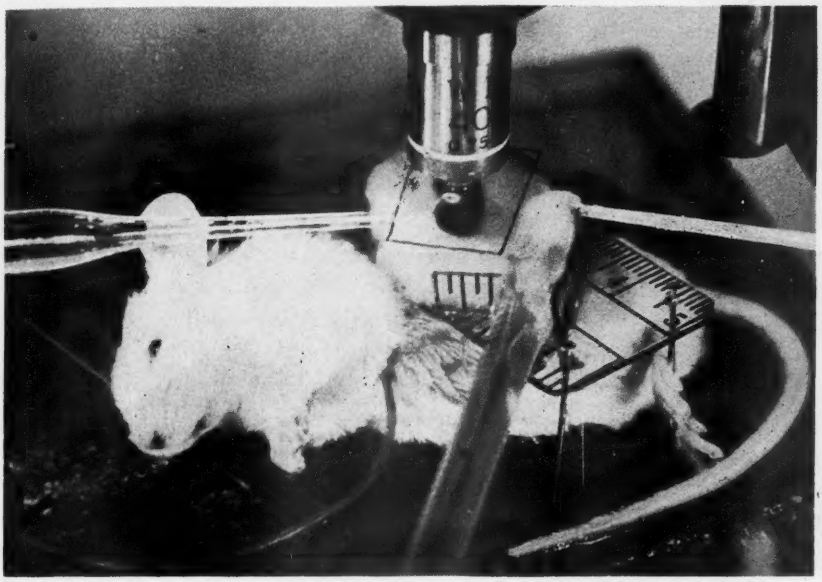


Fig. 3.—Transillumination apparatus arranged for observation of an exteriorized mouse spleen.

Hollow-tipped, fused quartz, illuminating rod enters chamber from left; immersion fluid delivery tube from right; and thermometer from foreground. Anesthesia fluid is being conveyed to this animal's left pectoral region. Lift from illuminator tip is directed upward, through spleen substance, into water-immersion objective.

(From MacKenzie, D. W., Jr., Whipple, A. O. and Wintersteiner, M. P.: Studies on the Microscopic Anatomy and Physiology of Living Transilluminated Mammalian Spleens. *Amer. Jr. Anat.*, v. 68, No. 3, May, 1941.)

and the second was the frequent displacement of the spleen with the movements of the animal when the sodium amytal anesthesia was repeated by hypodermic injection. By devising a celluloid supporting table for the spleen and by using a continuous clysis of sodium amytal solution these two causes of a shifting microscopic field were eliminated, and it became possible, for the first time, to observe areas of the vascular bed in the immobilized organ without any shift in the visual field over uninterrupted periods of several hours. Before this was done, the necessity of filling in gaps between the views of the vascular bed we feel was the cause of fundamentally wrong observations and conclusions (Fig. 3).

We regret, therefore, that we were unable to corroborate many of Knisely's findings and were compelled, as a result of a year of study, to differ

positively from him in his conclusions that the circulation is a closed circulation and in the three phasic functions he attributes to the venous sinuses. Our observations were made by at least three, and sometimes more, members of the Spleen Clinic and the anatomic components or structures of the vascular bed have been recorded by camera lucida drawings of the functioning fields studied by us over periods of one or more hours in the living transilluminated spleen of mice, rats, kittens and guinea-pigs.

I wish to say that by far the major part of this study of the vascular bed of the spleen, the improved method and technics, the continuous observations and the drawings, both free hand and camera lucida, were made by Doctor MacKenzie and by his able assistant, Mrs. Wintersteiner. The detailed report of this study by MacKenzie, Whipple and Wintersteiner[4] appeared in the American Journal of Anatomy, **68,** No. 3, 397–454, 1941.

The storm center in the controversy over open or closed circulation in the spleen has had to do with the question as to whether the arterial capillary terminates freely in the reticular meshes of the pulp spaces or directly into the venous sinuses. This question must be answered positively if certain essential points regarding the pathogenesis of many of the splenopathies are to be settled.

It is our definite conviction from our studies in the living spleen, which are in agreement with the conclusions of Mall,[10] Robinson,[11] Foot,[12] and Klemperer,[13] among others, from injected spleens and from microscopic sections, that the splenic pulp spaces provide the one and only link between the arterial and venous systems in the mammalian spleen. Individual pulp spaces are the most variable structures that we have observed in the spleen. For the most part they are tortuous, utterly irregular and inconstant channels, lined by fixed and wandering cells and by reticulum. They constitute a plexiform, three dimensional system of channels intimately connected with one another by actual or potential passages, fully adequate to transmit the cells as well as the fluid of the blood. The average width of the pulp spaces in the normal relaxed spleen of the mouse is six microns, but the diameter of a dilated pulp space has been measured to 16 microns. A collapsed or compressed space may have no visible lumen. On the arterial side of this fibrocellular sponge the pulp spaces communicate with the arterial capillary ampullae, or ampullae of Thoma. On the venous side, the spaces converge upon the stigmata, or free openings, of the venous sinuses and intralobular veins. Camera lucida drawings of this intermediary zone and of the pulp connections with arterial and venous systems are shown in Figures 4–7, and are in agreement with the semidiagrammatic drawing of the intermediary circulation in the human spleen (Fig. 8), as represented by Klemperer.[13]

Bear in mind this anatomy of the intermediary circulation in considering the pathology of chronic portal bed obstruction. The most characteristic feature in the microscopic sections of these splenomegalies is the widening and distention of the venous sinuses with a widening of the stomata resulting in a compression of the splenic pulp spaces. In addition, there is an hyperplasia

of the cytoplasmic reticulum. The back pressure in the venous sinuses, transmitted by the hypertension in the veins, causes the distention of the venous sinuses and the narrowing of the pulp spaces. This makes it more difficult for the blood to pass from the arterial capillaries into the pulp spaces. This results in hemorrhages about the trabecular arteries and at the periphery of the follicles, with a later development of nodular areas of fibrosis—the typical fibro-adenia described by Banti.

This partial exclusion of the splenic pulp from active circulation results in a gradual atrophy of the reticulum with connective tissue replacement and a fibrous spleen.

FIG. 4

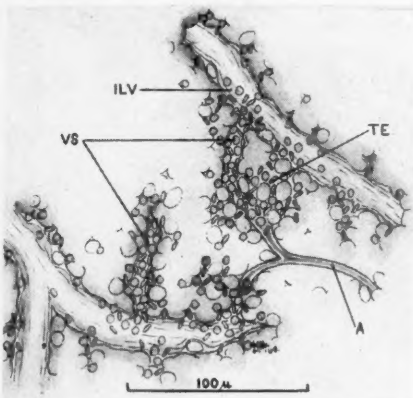
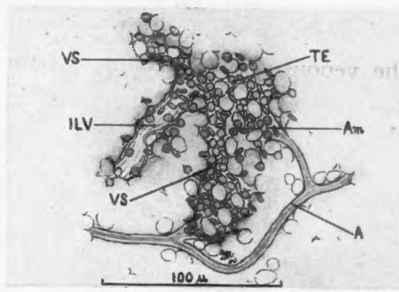


FIG. 5



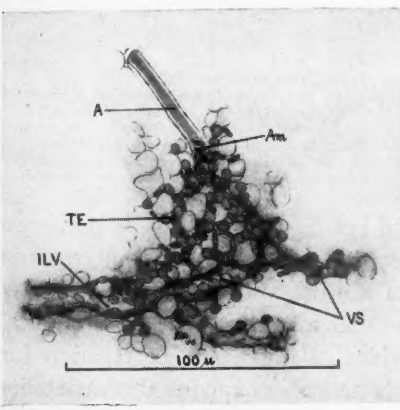


FIG. 6

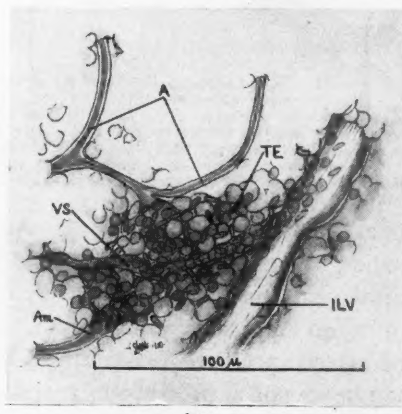


FIG. 7

FIGS. 4-7.—Intermediary circulation in transilluminated mouse spleens.
Arterial capillaries, A, are shown to communicate via ampullary dilatations, Am, with swamp-like pulp zones, through which erythrocytes trickle onward, *TE*, into venous sinuses, *VS*, and intralobular veins, *ILV*.
Camera-lucida drawings. Lens magnification of Figures 4 and 5, 400; of Figures 6 and 7, 600.
(From MacKenzie, D. W., Jr., Whipple, A. O. and Wintersteiner, M. P.: Studies on the Microscopic Anatomy and Physiology of Living Transilluminated Mammalian Spleens. *Amer. Jr. Anat.* v. 68, No. 3, May, 1941.)

OBSTRUCTIVE FACTORS

The amount of portal bed obstruction, the type and the site of obstruction, are all variable factors in individual patients with portal hypertension.

It is the discovery in the individual patient of these factors and their analysis that very largely determines the diagnosis, treatment and prognosis.

These patients with portal hypertension may be divided into two main groups: Group I. those having intrahepatic portal block; and Group II. those having extrahepatic portal block.

In the first group the cirrhoses, especially of the portal or Laennec type,

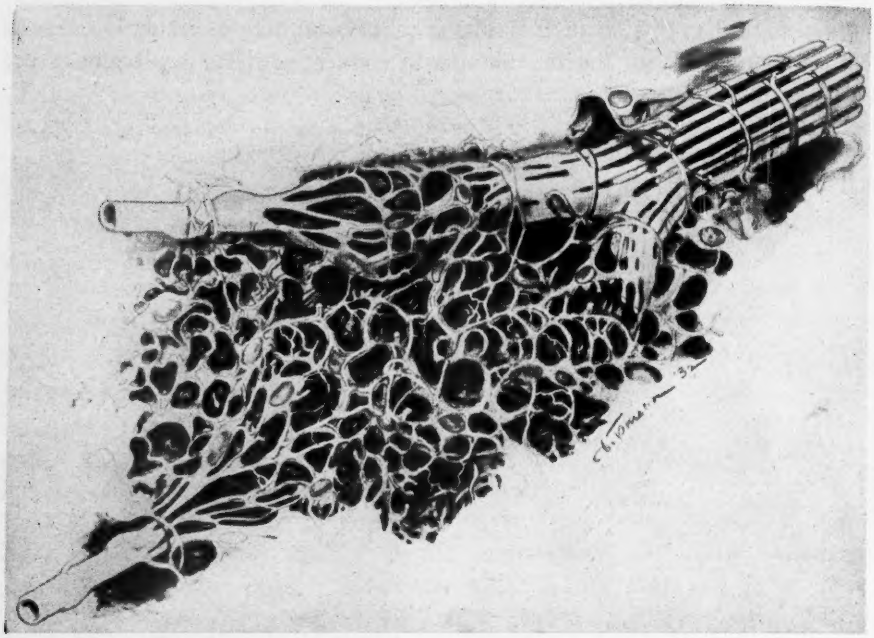


FIG. 8.—Semidiagrammatic drawing of intermediary circulation of human spleen.

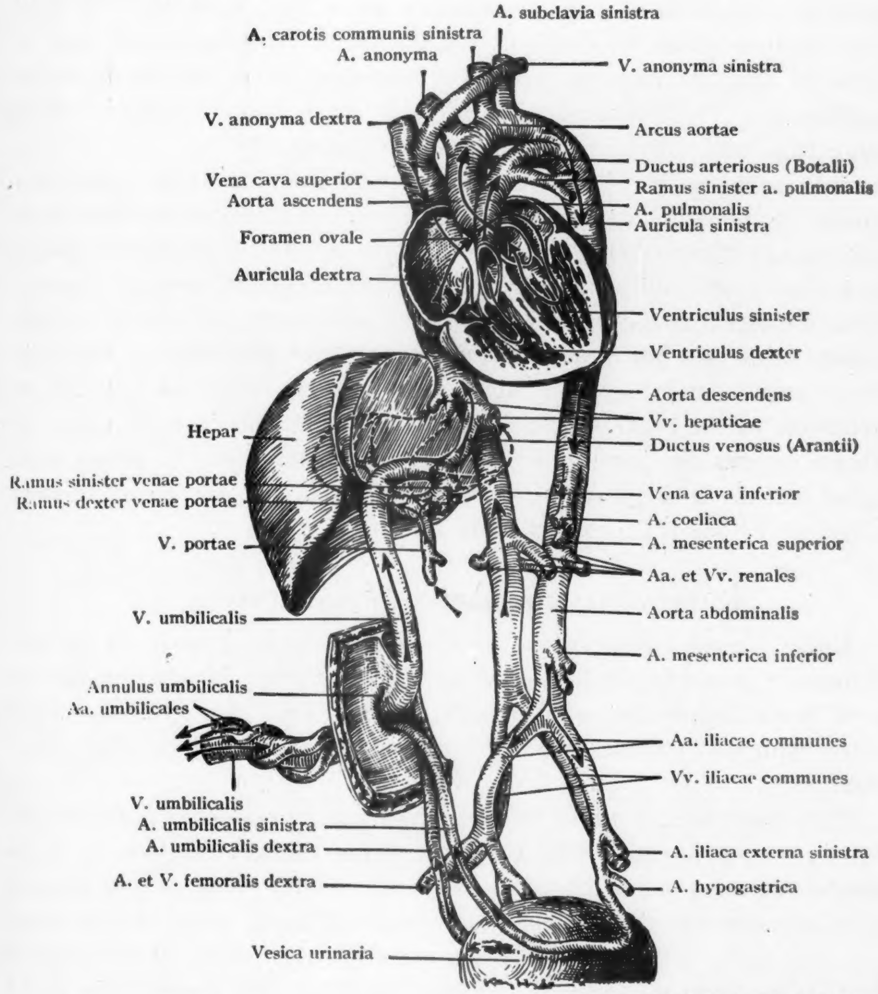
This drawing represents author's concept of architecture of reticulum and its relation to finest blood channels. Although it is not an actual reconstruction of serial sections, all structural details which are depicted in three dimensions have been observed histologically. For technical reasons, reticular fibers have not been drawn in different color but as ridges upon cytoplasmic syncytium.

(From Klemperer, P.: Chapter on the spleen in Downey's "Handbook of Hematology." Paul B. Hoeber, New York, 1938, p. 1633. This figure is reproduced by kind permission of the publisher, Mr. Paul B. Hoeber.)

are associated with portal block. As pointed out by Herrick[3] in his perfusion studies, there is a great difference between the ratios of the increase in portal vein and hepatic artery pressures of normal and cirrhotic livers, *i.e.,* 1 mm./40 mm., and 1 mm./6 mm. This mutual influence between the portal and arterial pressures within the liver provides an important explanation for the rise of portal pressure in portal cirrhosis and also explains the variability of portal hypertension in the cirrhoses and the presence or absence of gastro-intestinal hemorrhage, as a measure of portal hypertension.

The degree of portal hypertension varies in the different types of cirrhosis and in the patients with the same type of cirrhosis and varies in the same individual with cirrhosis at different times. Thus, we find some patients with portal cirrhosis without a history of gastro-intestinal hemorrhage and with no splenomegaly, others have repeated hematemeses and enlarged spleens with marked engorgement of collateral veins.

Group II. *Extrahepatic Portal Block.*—There are two types of obstruction seen in chronic occlusion of the portal vein and its main tributaries. The first is a replacement of the vein or its main tributaries with fibrous tissue or scar tissue with little or no canalization. The second is a transformation of the portal vein or its main tributaries or the enveloping tissue into a



From Callander's Surgical Anatomy
Philadelphia, W. B. Saunders Co.

FIG. 9.—Diagram of the fetal circulation about the time of **birth.**

cavernomatous mass of small tortuous vessels, a process spoken of as cavernomatous transformation of the portal vein.

In the first type of fibrous replacement there are two causative factors. The most common is an organization into scar tissue of a thrombosis of the portal vein or of a main tributary. The thrombosis may be the result of inflammation, trauma, or pressure from without by inflammatory or

neoplastic tissue. The second causative factor is an extension into the left portal vein or proximal to it into the main portal vein of the obliterative fibrotic process that takes place at birth in the umbilical vein and ductus venosus as they empty into the left portal vein (Fig. 9). This type of obstruction is fortunately rare but is seen in young children that begin at an early age to show portal block with splenomegaly and a Banti syndrome. In these cases the liver shows no cirrhosis, but the portal vein obstruction may be partial or complete, and the portal vein may show either fibrosis or partial canalization. The collateral circulation is marked and gastro-intestinal hemorrhage with splenomegaly is a common finding.

The pathogenesis of cavernomatous transformation is not definitely known. By some it is considered to be the result of an organized thrombosis with recanalization; others consider it to be the result of telangiectatic granulation tissue; while still others consider it to be a congenital anomaly (against this is the fact that evidences of portal hypertension do not appear in some patients with this lesion until adult life). Other pathologists, especially Pick,[14] argue convincingly that this is a neoplastic lesion, an angioma or cavernoma of the hepatoduodenal ligament, inasmuch as in some cases the process extends far beyond the limits of the portal vein. It would seem logical that the etiology of this lesion is a variable one, caused in different individuals by one of the above factors.

COLLATERAL CIRCULATION IN PORTAL BLOCK

Under normal conditions the portal pressure varies from 8 to 13 mm. of mercury, or 60 to 104 mm. of water. With increase in pressure, and as portal block distends the portal radicals, potential veins connecting the portal system with the systemic begin to appear and enlarge to by-pass the portal blood.

These have been classified by Pick[14] into two groups—the *"hepatopetale"* and the *"hepatofugale"*. When the circulation through the liver is unobstructed and the block is limited to the portal vein the blood may be shunted to a limited extent through the *hepatopetale* collateral veins, the so-called accessory veins of Sappey, which pass through the peritoneal covering of the liver, or from the stomach and pass either into the stem of the portal vein or into the substance of the liver, the so-called deep cystic veins, the epiploic veins of the gastrohepatic omentum, the hepatocolic and the hepatorenal veins, the diaphragmatic veins and the veins of the suspensory ligament of the liver.

In lesions causing intrahepatic block the *"hepatofugale"* circulation shunts a variable amount of the blood from the gastro-intestinal tract and spleen around the liver. McIndoe[15] has classified the various parts of the *"hepatofugale"* collateral circulation into three groups on an embryologic basis:

A. The veins located at the two sites of the gastro-intestinal tract where glandular epithelium unites with squamous epithelium, *i.e.,* at the cardia and at the anus. The veins at the cardia provide an outlet to the superior cava by way of the esophageal veins to the azygos system. The veins at the anus furnish an outlet to the inferior cava by way of the middle hemorrhoidal veins.

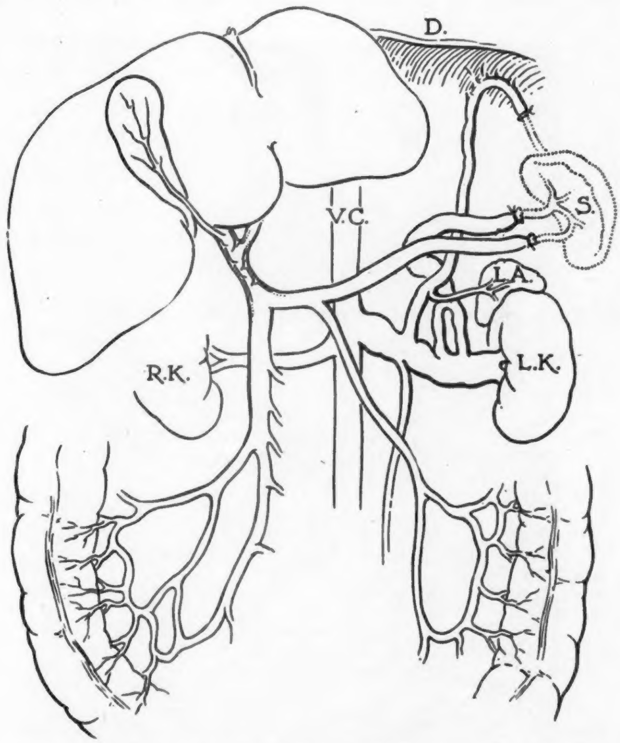


FIG. 10.—Diagram of the collateral circulation. The spleen(S) and the adjacent portions of the veins removed at operation are indicated by dotted lines. L.K. indicates left kidney; R.K., right kidney; V.C., vena cava inferior, and L.A., left adrenal gland. (From Simonds, *Archives of Surg.,* v. 33, 1936.)

B. The veins at the site of the obliterated fetal circulation, the paraumbilical veins in the round ligament of the liver.

C. The veins found at the sites within the abdomen where the gastrointestinal tract and its appendages, or the organs developed from it, become retroperitoneal developmentally, or adherent to the abdominal walls as a result of some pathologic process. These are sometimes spoken of as the veins of Retzius, and are well shown in the case reported by Simonds[16] (Fig. 10).

EXPERIMENTAL PRODUCTION OF CHRONIC PORTAL BLOCK

This can be done successfully only by a gradual process of obstruction. A sudden closure of the portal vein, or one of its main tributaries, such as

the splenic, results in acute engorgement with either death of the organ, or organs, drained by the portal or an atrophy of the part drained by the obstruction.

Chronic intrahepatic block has been successfully done in our laboratory by Rousselot and Thompson[17] by the repeated injection of finely divided particles of silica into the splenic or portal veins. This, in a period of 12 to 18 months, results in a lesion almost identical with the severe portal

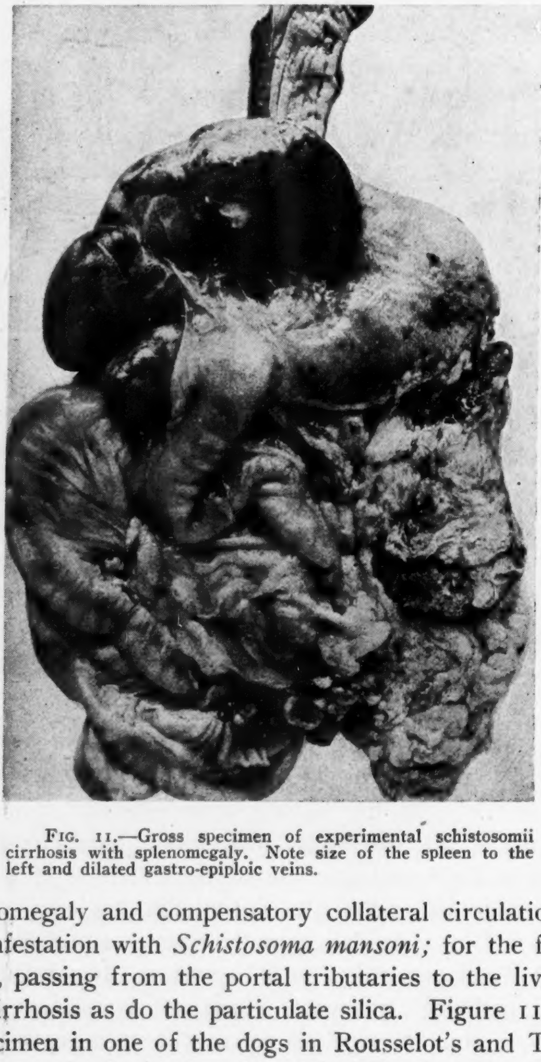


FIG. 11.—Gross specimen of experimental schistosomii cirrhosis with splenomegaly. Note size of the spleen to the left and dilated gastro-epiploic veins.

cirrhosis, splenomegaly and compensatory collateral circulation that is seen in victims of infestation with *Schistosoma mansoni;* for the fragmented ova of this parasite, passing from the portal tributaries to the liver produce the same kind of cirrhosis as do the particulate silica. Figure 11 is taken from an autopsy specimen in one of the dogs in Rousselot's and Thompson's experiments. These animals developed a typical Banti syndrome. Portal vein pressures in the animals showing cirrhosis and splenomegaly were recorded as high as 470 mm. of water. If allowed to live, some of these animals developed a massive ascites and showed tremendous esophageal varices.

Extrahepatic portal block is more difficult to produce experimentally.

The most successful chronic extrahepatic obstruction in our laboratory was accomplished by Rousselot and Rennie,[18] using cellophane bands placed around the portal or splenic veins, without shutting off the flow of the vein. The gradual production of dense scar tissue around the cellophane bands resulted in a chronic portal obstruction in about half of the animals.

THE SYNDROME OF PORTAL BED BLOCK

In many of the patients, and in experimental animals, portal bed block produces a fairly typical syndrome. Certainly, this is true of the cases showing a splenomegaly whether the block is intra- or extrahepatic. This syndrome consists of a variable secondary anemia, a leukopenia, a thrombocytopenia, a splenomegaly and a tendency to repeated severe gastro-intestinal hemorrhage, most frequently associated with ruptured esophageal varices. The liver may be cirrhotic or may be normal, depending upon the site of the portal bed obstruction (Fig. 12). This syndrome is frequently spoken of as Banti's syndrome.

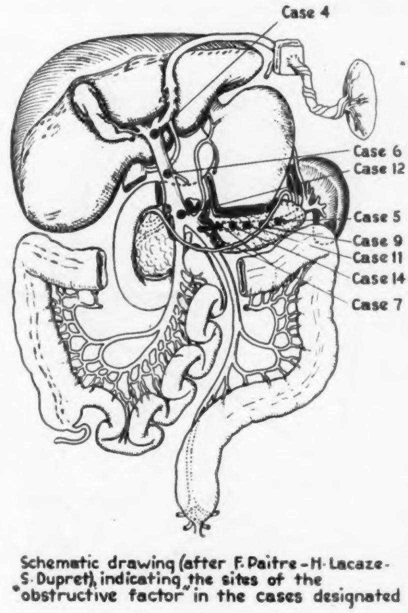


Fig. 12.—Sites of obstruction noted in cases from our clinic reported by Rousselot.

Banti,[19] in 1883, first called attention to this syndrome, and subsequently published a number of papers on the subject, maintaining that the hepatosplenopathy was caused by some unknown toxic agent that first caused the splenic enlargement and later produced a cirrhosis. He described this as a disease running a chronic course, progressing in three stages: (1) The anemic phase, with splenomegaly, asthenia and occasional gastro-intestinal hemorrhage; (2) the transitional stage, with oliguria, urobilinuria, hepatomegaly, brown discoloration of the skin and increasing gastro-intestinal bleeding; and (3) an ascitic stage, with atrophy of the liver, subicteric sclerae, hemorrhages from the mucous membranes and death from hemorrhage or liver insufficiency. He noted some of the hematologic findings, and described the pathology as present chiefly in the spleen and the liver, with almost constant findings in the splenic and portal veins. The pathognomonic lesion he described as a conspicuous thickening of the fibrillar reticulum around the central arteries of the malpighian corpuscles. "The fibro-adenic alterations in the follicle distinguish *morbus* Banti. Their absence rules out the disease."

In fairness to Banti, it must be said that the term Banti's syndrome has not been confined to the entity as described by him. On the other hand, so few cases of hepatosplenomegaly with the anemia, leukopenia and gastro-

intestinal hemorrhages, as described by Banti, show the three-stage process of the disease that the term Banti's syndrome has replaced to a large extent the term Banti's disease. Inasmuch as Banti insisted that the fibro-adenia of the follicles was the one pathologic finding in the spleen essential to the diagnosis of *morbus* Banti, the finding of this fibro-adenia in cases of splenomegaly, not fulfilling the clinical criteria of the disease described by him, and in cases with normal liver histology but with portal bed obstruction, invalidates the term Banti's disease.

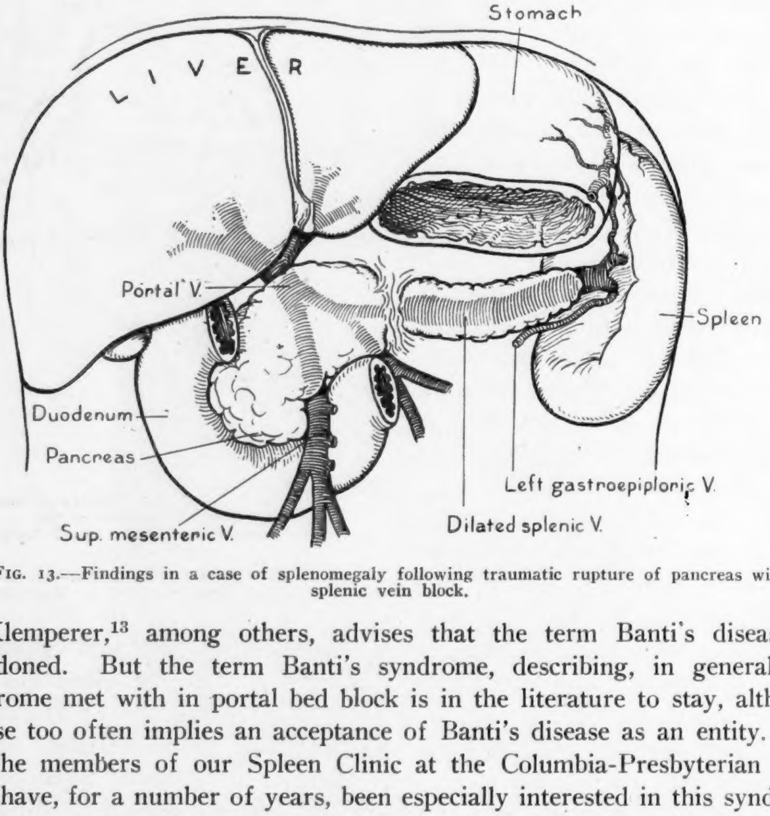


FIG. 13.—Findings in a case of splenomegaly following traumatic rupture of pancreas with splenic vein block.

Klemperer,[13] among others, advises that the term Banti's disease be abandoned. But the term Banti's syndrome, describing, in general, the syndrome met with in portal bed block is in the literature to stay, although its use too often implies an acceptance of Banti's disease as an entity.

The members of our Spleen Clinic at the Columbia-Presbyterian Hospital have, for a number of years, been especially interested in this syndrome of splenomegaly with anemia, leukopenia and thrombocytopenia, associated with the development of increased collateral circulation between the portal and peripheral venous systems and characteristic histologic changes in the spleen. The patient who first demonstrated to us this syndrome in complete conflict with Banti's hypothesis was a young, vigorous policeman giving the following history: In 1928, in attempting to stop a runaway team he was pinned between an elevator pillar and the tongue of the wagon. This resulted in a rupture of the pancreas, for which he was operated upon at the Post-Graduate Hospital by Dr. John Erdman. A few months later he developed a pancreatic cyst which Doctor Erdman drained. At this time his spleen was not enlarged and his blood picture was normal. During the next three years

he gradually developed a splenomegaly and an anemia with a leukopenia and thrombocytopenia, and had two severe hematemeses. The clinical picture and diagnosis of Banti's syndrome was evident. In 1932 I removed his spleen, finding, at that operation, a very large spleen with an enormously dilated splenic vein, which entered dense scar tissue at the site of the injury to the pancreas, resulting in splenic vein obstruction and diffuse collateral circulation between the splenic vein and branches of the left gastric and left gastro-epiploic veins. The liver appeared normal in every respect. This

TABLE I

SPLEEN CLINIC—PRESBYTERIAN HOSPITAL

Cases to March 15, 1945

	Cases With Splenectomy	Cases Without Splenectomy	Total No of Cases
Abdominal Buerger's disease	1	8	9
Anemia:			
Aplastic	2	84	86
Cooley's	9	17	26
Sickle cell	2	22	24
Banti—Extrahepatic:			
Cavernomatous transformation	4	2	6
Compression	2	3	5
Sclerosis of portal veins	5	1	6
Thrombosis	3	3	6
Stenosis of portal veins	3	1	4
Banti—Intrahepatic:			
Cirrhosis	36	59	95
Cirrhosis schistosomal	14	3	17
Banti—Obstructive factor undetermined	26	9	35
Boeck's sarcoid	2	6	8
Cyst of spleen	1	..	1
Gaucher's disease	10	3	13
Hemolytic jaundice:			
Atypical	10	11	21
Typical	53	20	73
Hodgkin's disease	2	172	174
Leukemia—Chronic myeloid	1	152	153
Normal splenectomies	42	..	42
Osteosclerotic myelofibrosis	3	14	17
Polycythemia	1	108	109
Purpura:			
Atypical	10	82	92
Idiopathic thrombocytopenic	52	26	78
Sarcoma of spleen	10	7	17
Splenomegaly— Undetermined origin	12	60	72
Total	316	873	1,189

patient made an uneventful recovery and his blood values promptly returned to normal and remained so for the next nine years of his follow-up. He had no recurrence of hematemesis.

This finding of a normal liver on the right and a splenomegaly on the left with an obstructive factor between and the picture of Banti's syndrome initiated certain studies which have clarified our ideas regarding the pathogenesis of what we agree with Larrabee[20] should be called congestive splenomegaly.

Our Spleen Clinic studies on splenic vein pressures made at the time of splenectomy and compared with peripheral venous pressures were reported by the Spleen Clinic in 1937.[21] These showed an increase of two to five times splenic vein pressure over peripheral venous pressure in cases presenting Banti's syndrome. Table II shows the comparative readings in the different types of splenomegaly. It is our present concept that Banti's syndrome is

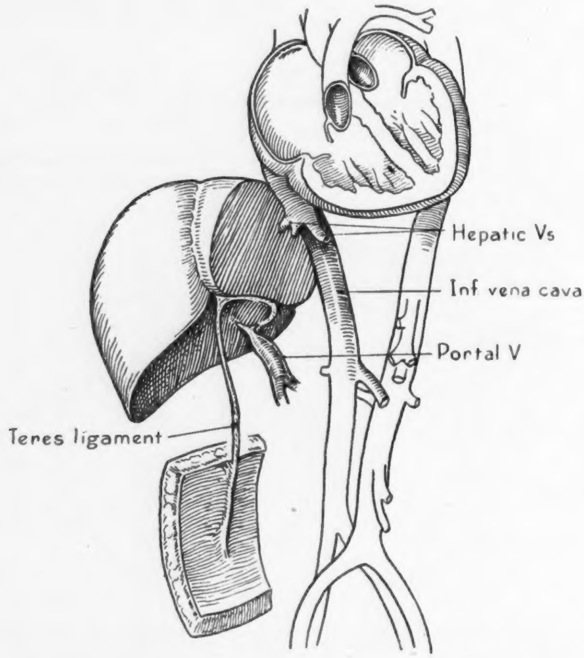


FIG. 14.—Autopsy findings in case of extrahepatic portal block following extension of obliterative process from the umbilical vein into the portal vein.

the result of mechanical obstruction to the flow of blood within the portal bed.

In the cirrhoses there is a variable amount of portal hypertension, determined by the amount of scar tissue in Glisson's capsule, the relation of the pressure in the hepatic artery to that in the portal vein and the extent of the hepato-fugal collateral circulation. For these reasons splenomegaly, gastrointestinal hemorrhage, leukopenia and thrombocytopenia are not always found in the cirrhoses. This syndrome is not characteristic of the biliary and cardiac cirrhoses.

On the other hand, in our experience, if the extrahepatic portal block, from whatever cause, is sufficient to produce a splenomegaly, Banti's syndrome is nearly always present, and a normal liver is usually found even in the cases of long standing, some of which we have followed for 10 to 20 years, and that because of recurrent esophageal hemorrhage have provided us with autopsy studies.

H. J. Ward: G-W. First admission: March 17, 1926-May 27, 1926. Twenty-third and final admission: July 22, 1939. Died: July 26, 1939.

Autopsy Report—Final Note: A young man, age 18, who had been in the hospital 23 times for repeated hematemeses, which first occurred at the age of 2.5 years. A diagnosis of Banti's disease was made at the age of five, and was followed by splenectomy. He developed extensive esophageal varices and these were cauterized several times, but

Table II

SPLENIC VEIN PRESSURE IN THREE CASES OF BANTI'S SYNDROME ASSOCIATED WITH CHRONIC SCHISTOSOMIASIS

Case No.	Splenic Vein Pressure Mm. H_2O	Simultaneous Arm Venous Pressure Mm. H_2O
1. P. R.	250	50
2. A. E.	335	105
3. G. P.	500	70

SPLENIC VEIN PRESSURE IN FIVE CASES OF BANTI'S SYNDROME ASSOCIATED WITH LAENNEC'S CIRRHOSIS

Case No.	Splenic Vein Pressure Mm. H_2O	Simultaneous Arm Venous Pressure Mm. H_2O
4. C. M.	275	12
5. G. M.	325	85
6. D. P.	450	125
7. L. DeR.	275	105
8. N. A.	470	140

SPLENIC VEIN PRESSURE IN THREE CASES OF TYPICAL HEMOLYTIC JAUNDICE

Case No.	Splenic Vein Pressure Mm. H_2O	Simultaneous Arm Venous Pressure Mm. H_2O
9. R. B.	105	80
10. N. B.	125	130
11. W. U.	120	85

intermittent bleeding persisted throughout his life. During the final few months a loud systolic murmur developed and the question of rheumatic endocarditis was raised. Terminally, his blood platelets fell sharply and he developed an hemiplegia which was thought possibly due to the formation of platelet thrombi.

Autopsy shows the cause for the splenomegaly and varices to reside in a greatly narrowed, trabeculated portal vein. Many collateral channels have been established. In some of these about the splenic vein as well as in the latter, and in the portal vein there is considerable phlebosclerosis. Hyaline thrombi are found in veins of the myocardium, kidneys, and rectum, and the right posterior cerebral artery is obstructed by a similar thrombus. The mitral valve bears large organizing vegetations, probably rheumatic in origin. The kidneys show what is probably an early intercapillary glomerulonephritis. There are also small foci of necrosis of tubular epithelium and beginning regeneration, the cause of which is unknown. Sections of the liver appear normal. There is no evidence of cirrhosis.

The pathogenesis of the portal vein lesion is open to discussion. It suggests cavernomatous transformation of a relatively simple type. An early, perhaps congenital, development of the lesion is indicated by the early onset of symptoms, and Doctor

Thompson suggests that the normal postnatal obliteration of the umbilical vein might conceivably extend into the portal trunk. The extensiveness of the collateral circulation, and the enlargement of the hepatic artery, are evidences of compensation in a growing individual.

THE DIAGNOSIS OF PORTAL HYPERTENSION

Patients with a portal hypertension great enough to cause an enlarged spleen usually present the leukopenia and thrombocytopenia and secondary anemia of Banti's syndrome. There may be no history of gross gastro-intestinal hemorrhage. The differential diagnosis from other splenomegalies is largely determined by accurate hematologic studies.

The site of the portal block, as to whether it is intrahepatic or extrahepatic can usually be determined by certain liver function tests. If there is a high retention of bromsulphalein in the blood 30 minutes after intravenous injection, if the hippuric acid test is positive, if there is a reversal of the albumin-globulin ratio or if the cephalin flocculation test is positive, the presence of a cirrhosis with intrahepatic portal block is fairly certain. On the other hand, if these tests are negative it is safe to assume that the block is extrahepatic. But this does not necessarily determine the site of the extrahepatic block. In patients with normal liver function if there is a history of an antecedent pancreatitis or a severe trauma to the epigastrium the diagnosis of a splenic vein thrombosis is a valid one. In a young child with normal liver function tests, giving a history of hematemeses at an early age the diagnosis of portal vein occlusion as a result of continuation of the obliterative process in the umbilical vein and ductus venosus is a probable one.[22] The final determination of the site of the extrahepatic block in many patients can be made only at the autopsy table, for the dissection necessary to demonstrate such a block is neither safe nor feasible in the great majority of patients on the operating table. We have been unable to determine the site of the extrahepatic block at the time of splenectomy in more than half of our patients, although in our more recent operations we have demonstrated the block by diodrast venograms taken at the time of determining portal vein pressures with roentgenograms at the operating table.

THE TREATMENT OF PORTAL BED BLOCK

The therapy of the cirrhoses not associated with portal hypertension does not come within the scope of our topic. It is the portal bed block, both intra- or extrahepatic, associated with Banti's syndrome that poses the problem to be considered in this discussion.

Three factors, the site of the block (Fig. 12), the degree of portal hypertension and the extent and competency of the collateral circulation determine the size of the spleen and the incidence of gastro-intestinal bleeding. The two latter components of the syndrome are the usual indications for attempted surgical therapy. In the past, three lines of surgical attack have been fol-

lowed—by splenectomy; by the establishment of collateral circulation with intra- or extraperitoneal omentopexy; and by ligation of the tributaries to esophageal varices. These will be considered separately.

The spleen carries a very large load of the portal blood, estimated at 40 per cent of the total. Splenectomy, especially if the spleen is as large as it usually is with Banti's syndrome, removes not only a large area of the portal bed, but shuts off many of the large collateral veins in the gastrolienal ligament that feed into the varices around the diaphragm, the cardia and the esophagus.

If the portal block is in the splenic vein the removal of the spleen results in a permanent cure, with the disappearance of the Banti syndrome. Unfortunately, this site, in the splenic vein, for portal bed block is not a common one. We have had only five such cases, but they were all cured, with no recurrence of hematemesis. But even with the block in the main portal vein splenectomy provides relief for a variable time because of the removal of a large area of portal bed and until the portal hypertension builds up again.

The establishment of an adequate collateral circulation by omentopexy: whether by the Talma-Morison procedure of placing the omentum in contact with an abraded or irritated surface of the liver or by suturing the omentum in contact with the split rectus muscle, the efficacy of omentopexy is questionable. In our experience, if the operation is done in the presence of a well-established collateral venous circulation in the abdominal wall, as evidenced by dilated superficial veins, or as shown by infra-red photographs, the results in a few cases are encouraging, but probably due to Nature's efforts rather than to the surgeon's.

Attempts to ligate the tributaries feeding into the veins of the cardia and esophageal varices have been very disappointing. Nor have the injection and coagulation methods to obliterate the esophageal varices been any better. At best, these procedures shut off one of the chief collaterals between the portal and systemic circuits and increase the portal hypertension. The large number of patients with portal vein block and Banti's syndrome whose spleens we have removed, but who continued to have recurrent gastro-intestinal hemorrhage, challenged the members of our Spleen Clinic to seek a more effective and permanent therapy.

Our efforts to anastomose branches of the mesenteric veins to the spermatic, the ovarian and the inferior cava by suture technic had failed. These suture anastomoses dealing with small veins are technically difficult and even with anticoagulants usually close by thrombosis. Attempts to make such shunts in the past have been reported by Gunn[23]—right ovarian to the portal; Villard and Tavernier[24]—ovarian to the superior mesenteric; and Meursing[25]—spermatic vein to the splenic, without success. Bogorts[26] anastomosed the superior mesenteric to the inferior cava and reported a good result. One

month later the spleen had decreased in size and ascites had disappeared. There was no later follow-up note.

We had discussed the more extensive procedures for portacaval shunt based on the principle of the Eck fistula, but because of the difficulties of dealing with the engorged portal tributaries and the disadvantages of suture anastomosis, with the threat of thrombosis, we made no attempts to carry out such procedures until Blakemore, of our Surgical Staff, developed his endothelial-lined vitallium tube nonsuture technic for bridging large vessel defects. Blakemore and Lord[27] have recently described this technic, and have made a major contribution to vascular surgery. Before describing this technic as applied to the problem of portacaval shunting operations, it is pertinent to discuss the history of portacaval anastomosis both in experimental animals and in human cases.

Nikolai Vladimirovich Eck was a Russian physiologist, born in 1847. In 1877, he published his report on "The Ligature of the Portal Vein."[28] He developed the Eck fistula for the experimental study of diseases of the liver and the relation of the liver to metabolism. Eck suggested that a portacaval fistula might be used to sidetrack obstruction in the portal vein, but the procedure found no trial for many years, until Tansini,[29] in 1902, advocated it on the basis of his animal experiments.

Vidal,[30] of Angers, claimed to have done this operation for the first time upon a patient with portal obstruction in 1903. This patient lived 14 weeks, and died of a septic endophlebitis. De Martel[31] reported a patient upon whom he had done an Eck fistula in 1910. The patient succumbed shortly with anuria. Lenoir,[32] according to Rosenstein, carried out an end of portal to side of cava anastomosis, but the patient died of anuria. Rosenstein[33] presented a 60-year-old woman, before the 41st Congress of German Surgeons, upon whom he had done an Eck fistula for cirrhosis and ascites in 1911. She had been tapped repeatedly, and five months after the Eck fistula operation this patient required only an occasional paracentesis with much less ascites.

The Eck fistula has been carried out in experimental animals very many times. Probably no one has had so much experience with constructive analysis in the study of Eck fistula dogs as George H. Whipple. His recent report[34] on his study of Eck fistula animals is an amazing example of carefully analyzed data on the metabolism of hemoglobin and protein in normal and in depleted Eck fistula dogs. He has observed some of these animals for periods as long as eight years, maintained in apparent state of health. They appear normal in all respects, activity, appetite, digestion and weight, but occasionally they may show increased thirst, diuresis, a trace of jaundice or lack of appetite and vague intoxication, evidences of disturbed protein metabolism.

In a letter,[35] written to me recently, Dr. Whipple says that the Eck fistula in dogs consists of a large opening between the portal vein and vena cava, usually 12 mm. in long diameter. The portal vein is ligated and crushed

just as it enters the liver. All operations are checked at autopsy to show that the fistula actually was present during the period between operation and death. Unless the portal vein is ligated the side-to-side opening will promptly close. In this series, Dr. Whipple says the hepatic artery is adequate to support relatively normal clinical activity for many years. He states that he has no knowledge of observations on human Eck fistulae, and considers the operation in cases with portal hypertension an extraordinarily interesting field for protein metabolism studies. He predicts that, as in experimental animals, they will have periods of good and periods of poor protein production.

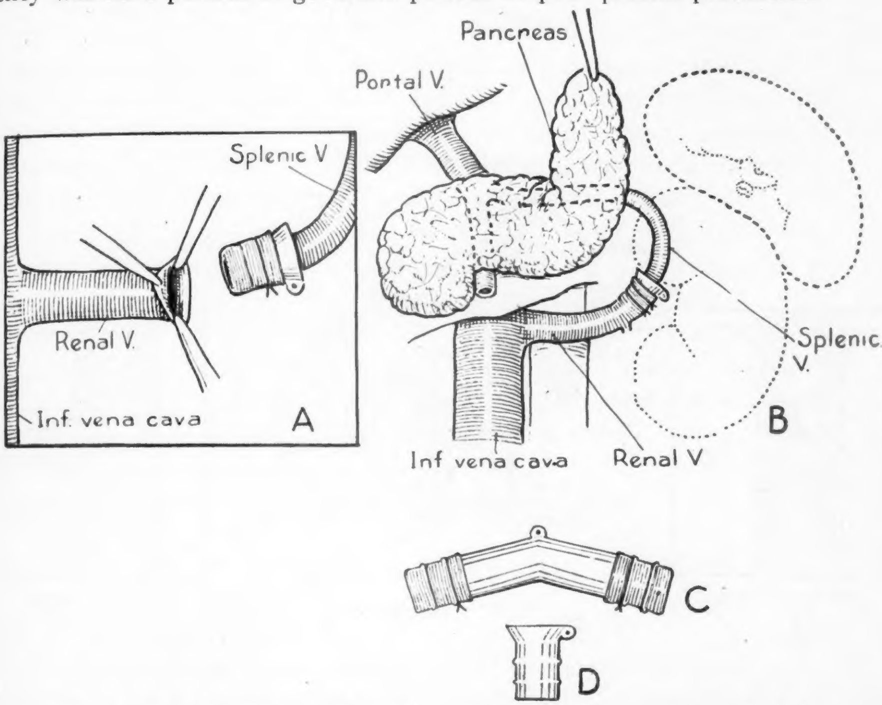


FIG. 15.—Showing technic employed in use of vitallium tubes in portacaval shunts.*

Quierolo,[36] in 1893, carried out a procedure for an Eck fistula in 16 dogs which was never given adequate recognition. He everted the cut-end of the portal vein over a glass tube and introduced this into an opening in the inferior vena cava, below the renals, taking care not to have the ligature tying the everted portal enter the cava. Two of these animals lived six months after operation. We were not aware of Quierolo's work until we had completed portacaval shunts in six of our patients.

Blakemore had demonstrated, in a large number of animal experiments and in several clinical cases, that arteriovenous anastomosis, arterio-arterial anastomosis and vein graft anastomosis could be done successfully by his nonsuture method in blood vessels of the extremities. By everting the end

* For details of this procedure see Figure 2 in article by Blakemore and Lord on page 479 of this issue of the ANNALS OF SURGERY,

of a vein or artery over the end of a short funnel-shaped vitallium tube, the endothelial surface can be introduced into the open end of another vein or artery to maintain blood flow, with avoidance of thrombosis because of intact endothelial lining. Or by using such tubes at either end of a vein graft vascular defects in arteries or veins can be effectually bridged (Figs. 15–16). Using this principle, Dr. Blakemore and I have carried out ten of these major procedures, five consisting of uniting the splenic vein and left renal veins, after

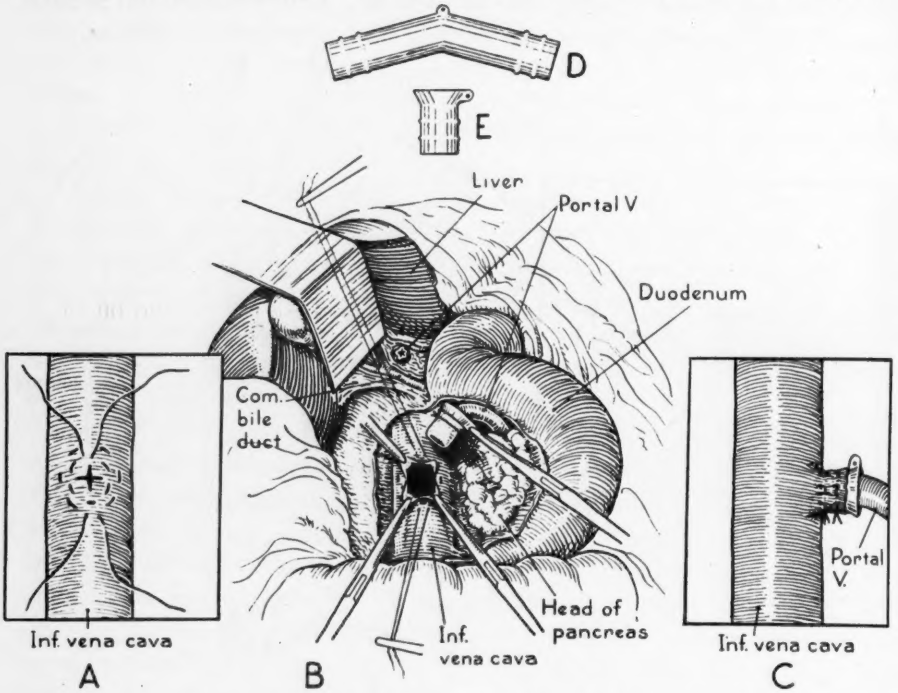


FIG. 16.—Showing technic employed in use of vitallium tubes in portacaval shunts.*

removing the spleen and left kidney. In our last five patients we have anastomosed the portal vein to the inferior cava, end-to-side. All these patients have survived their operations.

These procedures are as yet purely experimental. They have been carried out in patients that had had repeated severe hemorrhages, and for whom conservative measures offered no hope. The results in five of these patients have shown such a marked improvement in their liver function tests and disappearance of ascites or hemorrhage that we have been encouraged to continue, with improved technic, our efforts to provide an adequate short-circuiting of portal blood by the nonsuture technic. These are difficult, trying operations in the presence of engorged portal radicals, and require the teamwork of surgeons experienced in upper abdominal and in the basic principles of blood vessel surgery. Present-day methods for preventing and com-

* For details of this procedure see Figure 3 in article by Blakemore and Lord on page 481 of this issue of the ANNALS OF SURGERY.

bating shock; for improving compromised blood clotting mechanism and hypoproteinemia; together with the Blakemore nonsuture technic account for the fact that all ten of these patients survived the critical operative and postoperative period. It will require a follow-up period of three years, or more, to determine the value of these portacaval short-circuiting procedures. At least they represent a bold attempt to deal with the problem of portal hypertension in its life-threatening forms.

Four other splenorenal vein anastomoses for portal hypertension have recently been performed by Dr. Alfred Blalock,[37] who writes me that two of these patients have had a disappearance of ascites and are remarkably improved. On the other hand, he says his enthusiasm is somewhat curbed because the other two patients have died since operation from recurrent bleeding from esophageal varices. He thinks this may be due to occlusion of the anastomosis as a result of the attendant trauma at time of the operation.

With the present improved measures of preventing parenchymal liver damage with high protein-carbohydrate diets, high vitamin therapy, the lives of patients with cirrhosis are prolonged, but many of these patients go on to the development of portal hypertension and gastro-intestinal bleeding. It is also evident that the large number of men in the armed forces invalided by damaged livers, the result of infectious hepatitis, will become an increasing problem with the development of portal cirrhosis. The problem of therapy for hemorrhage in cirrhosis will continue to be a serious one. Doctors Blakemore and Lord are publishing a detailed report on the technic of portacaval anastomosis in this number of the ANNALS OF SURGERY.

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THE TECHNIC OF USING VITALLIUM TUBES IN ESTABLISHING PORTACAVAL SHUNTS FOR PORTAL HYPERTENSION*

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Ever since Nikolai V. Eck,[1] a Russian physiologist, successfully performed experimental anastomosis of portal vein to vena cava, in 1877, surgeons have been interested in its clinical application for the relief of portal hypertension. The rare reported instances[2-7] of attempts at the establishment of portacaval shunts by suture, and the by-and-large discouraging results, attest to the technical obstacles to its clinical accomplishment. The technical simplicity and efficiency of the nonsuture method of blood vessel anastomosis using vitallium tubes when employed in the anastomosis of arteries[8] lead to its experimental and clinical trial in the establishment of portacaval shunts.

Basic differences in the hemodynamics of the venous systems *versus* arterial so exaggerate the importance of certain technical aspects in the performance of anastomoses as to make it seem worth while to discuss in some detail the adaptation of the nonsuture method using vitallium tubes to the establishment of portacaval shunts.

At the outset, a general statement may be made, namely, that technical details conceded to be important to the success of arterial anastomosis must be executed with even more meticulous care to insure the success of portacaval shunt anastomoses. The purpose of uniting the portal and caval systems is to reduce portal hypertension and thereby lessen the tendency to gastrointestinal hemorrhage and the formation of ascites. This being true, a shunt capable of handling a large volume of blood should be established.

SPLENORENAL ANASTOMOSIS

This type of portacaval shunt is capable of handling a large volume of blood and, in addition, has the peculiar advantage of eliminating a sizable portion (estimated at 40 per cent) of the total circulating portal blood volume by splenectomy. So far our clinical experience has been limited to end-to-end anastomosis of the splenic vein to the left renal vein using the nonsuture vitallium tube technic. But, the facility with which an end-to-side anastomosis may be carried out using a vitallium tube affords an alternate method to the sacrifice of the kidney.

TECHNIC

The spleen is mobilized. In these cases of congestive splenomegaly it is unnecessary to emphasize that extreme caution must be exercised in the control of hemorrhage during mobilization. The vasa brevia are ligated with transfixion sutures of No. 1 Deknatel and the gastrosplenic omentum cut through. Next, the tail of the pancreas is separated from the splenic pedicle

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and the phrenicolic ligament is cut. This now permits freedom of movement of the spleen for a better examination of the splenic vein in the region of its bifurcation.

It is absolutely essential that the full length of the splenic vein be preserved with minimum trauma during splenectomy. In cases with large spleens in which there is persistent bleeding from disrupted adhesions it becomes

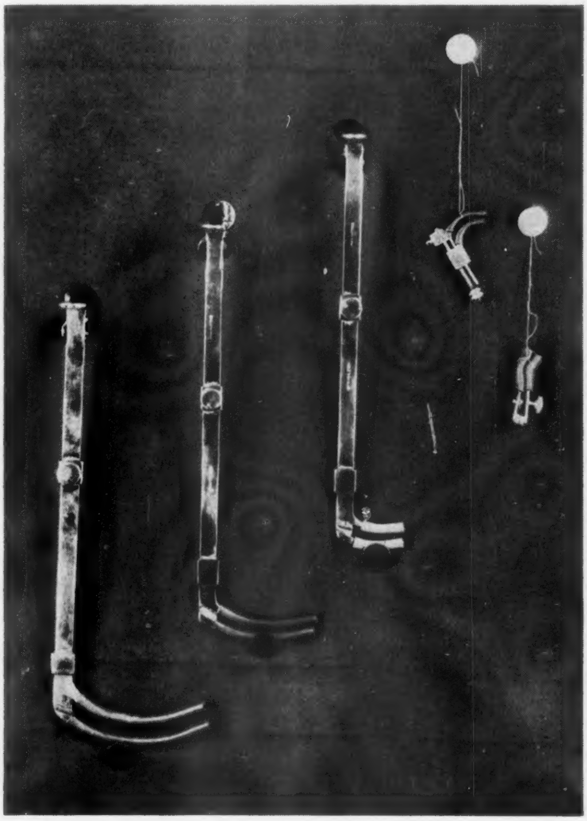

FIG. 1.—Clamps designed by Dr. Armistead Crump for the control of blood flow during the performance of portacaval anastomoses. The two large clamps are suitable for occluding the vena cava. The long handle clamps are easily applied to deep-seated vessels.

necessary to control the splenic artery at once by section between transfixion ligatures of No. 3 Deknatel silk. Otherwise, it may seem wiser, after isolating the artery at the chosen site, to defer ligation until most of the smaller splenic vein branches have been ligated by transfixion ligatures of B and C Deknatel silk. Following ligation of the splenic artery, the spleen may be somewhat emptied of its blood and the splenic vein ligated at once just at its distal primary branching. The blood is then milked far proximalward in the vein and a rubber-shod clamp applied. The smaller rubber-shod clamps illustrated in Figure 1 are handy for this purpose. Immediately following removal of the spleen the stump of the splenic vein should be opened, triangu-

lated with mosquito clamps and thoroughly irrigated with normal saline using a blunt-end syringe.

To effect a comfortable anastomosis of the splenic vein to the left renal vein it is best to mobilize the splenic vein for a distance totaling eight, or more, centimeters. To do this necessitates careful ligation of small pancreatic branches. These branches should be ligated flush with the splenic vein with C Deknatel silk then clamped distally and cut.

A transperitoneal approach is made to the left kidney with retraction of the descending colon medially. The kidney is mobilized and any accessory vessels ligated and cut. The ureter is identified at the lower pole of the kidney, ligated with No. 1 Deknatel silk, clamped proximally and cut. The renal artery is carefully isolated from the vein and ligated at a comfortable site using a transfixion ligature of No. 3 Deknatel. The branching of the renal vein is then studied to make sure that a maximum length of the main renal vein may be preserved for anastomosis. The renal vein is dissected back from the kidney for a distance of five or six centimeters. A rubber-shod clamp is applied as far proximally as possible. The renal artery may then be clamped just distal to the transfixion ligature, cut and again ligated. The vein is sectioned as close to the kidney as possible and the latter removed. Immediately thereafter the stump of the renal vein is triangulated with mosquito clamps and thoroughly irrigated with normal saline.

The splenic vein stump is irrigated with normal saline. A proper-sized vitallium tube (Fig. 2, D) is selected. A tube too large for the vein will present a funnel-like narrowing of the latter after mounting (cuffing). The end of the vein is passed through the tube, triangulated with mosquito clamps and everted (cuffed) over the end of the tube. The vein is held in place by a ligature of No. 1 Deknatel silk placed behind a holding ridge (Fig. 2, A). The tube-mounted splenic vein is freshly irrigated and then wrapped with vaselined gauze.

The renal vein stump is properly trimmed and then triangulated with mosquito clamps. A No. 3 Deknatel silk ligature is laid loosely about the vein. The vitallium tube is grasped with a holding clamp and the intima-covered end is directed toward the renal vein stump. Care is taken to see that neither vein is twisted. They are freshly irrigated with saline, following which the vein-covered end of the vitallium tube is introduced into the renal vein so that the latter comes up well proximal to the tying (holding) ridge on the tube (Fig. 1, A). The previously placed silk ligature is then tied very tightly about the renal vein, approximating it to the splenic vein at a point proximal to the tying ridge upon the tube. A second ligature of No. 3 Deknatel is placed in identical manner. Surgeons knots are essential for the maintenance of the necessary tension in these holding ligatures. Finally, a ligature of No. 1 Deknatel is tied, just snug, about the renal vein approximating it to the splenic vein near the end of the tube. The latter is most important as it keeps blood from penetrating between the two intimas. Figure 1, B shows completed anastomosis. It is arranged to release the rubber-shod

clamp on the splenic vein to be immediately followed by release of the rubber-shod clamp on the renal vein. The distended splenic vein should curve gently to its junction with the renal. Any tendency to acute angulation should be corrected. As much peritonization of raw surfaces as is possible should be carried out and careful ligation of any bleeding vessels before closing the abdomen.

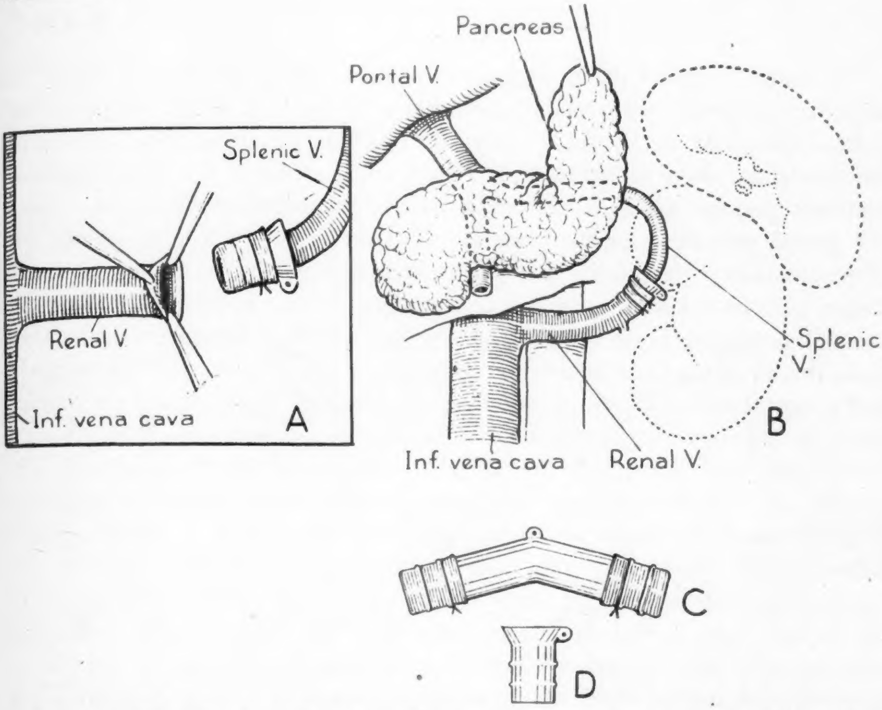

FIG. 2.—A. Illustrating the method of everting the renal vein for the introduction of the vitallium tube bearing the splenic vein. The clamp on the flanged portion of the vitallium tube for its guidance and the rubber-shod clamps upon the splenic and renal veins have been omitted. B. is a semidiagrammatic sketch of the completed anastomosis. Note the placement of the ligatures upon the vitallium tube. C. A vitallium tube with a vein graft mounted. D. An improved design of vitallium tube for end-to-end or end-to-side splenorenal anastomosis. There are two tying ridges placed 2 and 4 mm., respectively, from the end. Note the tab on the flange for the application of a holding clamp.

It is undesirable, for several reasons, to resort to the use of a vein graft in the performance of a splenorenal anastomosis. Though tension and angulation must be avoided, it is our opinion that the use of a graft is rarely necessary providing an adequate length of splenic vein is painstakingly mobilized. In our experience, it was necessary to resort to a vein graft in only one out of five cases. In this case, because of the unusual turgidity of the intervening tissues, due to extreme edema, the splenic vein did not adequately reach the stump of the left renal vein though it apparently had been mobilized over a length of approximately eight centimeters. The gap was bridged using a segment of superficial femoral vein. In the rare case in which the use of a vein graft is indicated, it is our belief that it is best (though this point has not been proven) to employ a vein-lined vitallium tube. Figure 1, C illustrates a vitallium tube, satisfactory in design, for this purpose.

ANASTOMOSIS OF THE PORTAL VEIN TO THE VENA CAVA BY THE NONSUTURE METHOD

The Eck fistula type of portacaval shunt has the advantage of size. An end-to-side anastomosis of the portal vein to the vena cava by the nonsuture vitallium tube technic affords an estimated blood carrying capacity 30 to 40 per cent greater than a splenorenal anastomosis.

TECHNIC

In order to avoid the undesirable use of a vein graft it is necessary to mobilize the portal vein from its bifurcation at the liver to the origin of the splenic vein. At the outset it is best to mobilize the descending portion of the duodenum along its lateral wall. This, with cutting of the hepatoduodenal ligament permits adequate retraction of the duodenum medialward. Since the portal vein lies slightly posterior and medial to the common duct, the above maneuver facilitates medial displacement of the common duct and, hence, permits a lateral approach to the portal vein. Entering the abdomen through a transverse or a right rectus incision with a lateral extension does have the advantage of facilitating a combined lateral and anterior approach to the portal vein. However, whether the approach be combined or anterior only, the common duct is mobilized sufficiently to swing it out of harm's way. The portal vein is carefully mobilized by sharp and blunt dissection. The placing of an umbilical tape or a small Penrose tube about the vein with gentle traction facilitates its dissection. The cystic vein is ligated with C Deknatel silk flush with the portal vein, clamped distally and cut. If the pyloric vein is found at or a few millimeters proximal to the origin of the splenic vein, it may be spared, otherwise it is ligated with C Deknatel silk and sectioned. A rubber-shod clamp is placed on the portal vein at the origin of the splenic vein. A transfixion ligature of No. 3 Deknatel silk is placed around the portal vein at its bifurcation close to the liver, care being taken not to injure the hepatic artery or common duct. The vein is transected four millimeters distal to the ligature. The portal vein is finally irrigated thoroughly with normal saline using a blunt-nose syringe and covered with a moist abdominal pad.

The vena cava is carefully mobilized by combined sharp and blunt dissection from the level of the liver down past the entrance of the left renal vein to the upper level of the right renal vein. The early passage of an umbilical tape about the vena cava with gentle traction serves to facilitate the dissection. Several small vein branches will be encountered posteriorly that will necessitate ligation with C Deknatel silk. A large rubber-shod clamp (Fig. 1) is placed about the vena cava at the upper level of the left renal vein but is not tightened to occlude the vessel.

The portal vein is now passed through a proper-sized vitallium tube. The end of the vein is triangulated with mosquito clamps. The tube is held firmly by a clamp, the end of the portal vein is then everted (cuffed) over the end of the vitallium tube. The vein is held in place by a No. 1 Deknatel

ligature tied tightly behind a tying (holding) ridge upon the tube, using a surgeon's knot (Fig. 3). The vein-covered vitallium tube is now swung out from behind the common duct, over the vena cava and a site for the anastomosis is selected. It is most important to select a site that will not result in angulation or compression of the portal vein. The portal vein is again irrigated with normal saline, covered with vaselined gauze to protect the exposed intima and again returned to its former position.

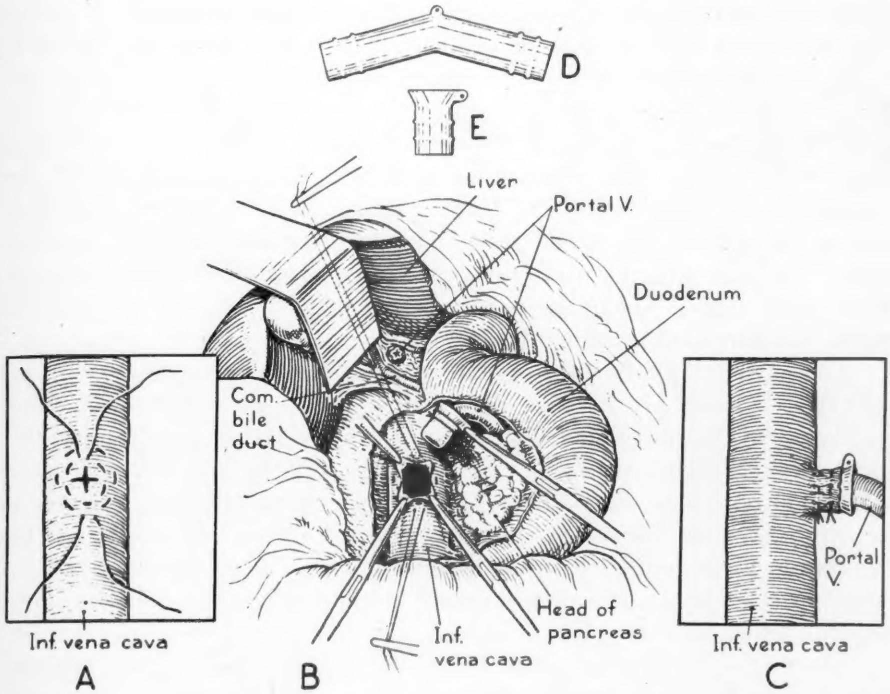

FIG. 3.—A. Illustrating placement of the purse-strings in the vena cava and the centering of the cruciate incision for implantation of the vitallium tube bearing the portal vein. B. shows the tube bearing the portal vein about to be introduced through the opening in the vena cava. C. The completed anastomosis. Note the vena cava wall is drawn well up on the vitallium tube. D. A tube suitable in design for vein graft bridging. E. A late design double-ridge tube with a holding tab.

The area of anterior vena cava wall selected as the site for the anastomosis is cleared of adventitia. New, thoroughly tested No. 3 Deknatel silk, threaded on a small Ferguson needle is introduced as a purse-string in the vena cava wall (full-thickness) at the site chosen for the anastomosis. The silk should be well vaselinized before its introduction. The purse-string is introduced to form a circle the diameter of which is four millimeters larger than the diameter of the vitallium tube selected for the anastomosis. A second circular purse-string, starting at the opposite side (Fig. 3, A) is placed two millimeters outside of the first one.

A second rubber-shod clamp is put in position about the vena cava as close to the liver as possible. The distal rubber-shod clamp is now quickly tightened to completely occlude the vena cava at the upper level of the left

renal vein, followed by tightening of the proximal rubber-shod clamp. The time at which the occlusion is made is noted and recorded. A cruciate incision through the vena cava wall, not exceeding in length the diameter of the vitallium tube, is exactly centered within the inner purse-string area. The apex of each quadrant of vena cava wall thus formed is grasped with mosquito clamps and the vena cava irrigated, using several syringefuls of normal saline.

The first turns of a surgeon's knot are placed in each purse-string but not tightened. The vitallium tube bearing the portal vein is grasped with a clamp and advanced through the opening into the vena cava against steady counter traction upon the mosquito clamps (Fig. 3, B). Care must be taken to see that the vitallium tube is not rotated to produce twisting of the portal vein. The vena cava is pulled well up on the vitallium tube so that the inner purse-string, as it is tightened, will fall proximal to the tying (holding) ridge on the tube. The purse-string is finally drawn very tight and the surgeon's knot completed. The holding clamp on the vitallium tube may now be removed and the second purse-string tied just snug about the vitallium tube. The latter will, if correctly placed, tighten about the tube just proximal to the distal ridge. The mosquito clamps may now be removed. Figure 3, C shows the completed anastomosis.

To establish blood flow through the anastomosis first release the proximal rubber-shod clamp on the vena cava. Next release the rubber-shod clamp on the portal vein to be immediately followed by release of the distal rubber-shod clamp on the vena cava. Duration of occlusion of the vena cava is noted and recorded. Finally, the portal vein is inspected for angulation or constriction. Any change of position is noted during the return of the duodenum to its normal position. Omentum may be placed over any unperitonized surfaces. Hemostasis should be checked and the abdomen closed carefully in layers.

Should the use of a vein graft to complete the anastomosis be unavoidable, the external iliac vein more nearly approximates the diameter of the portal vein and only a short segment (6–7 cm.) is required. Figure 3, D illustrates a satisfactory design of a vitallium tube to be lined by a vein graft.

SELECTION OF CASES FOR PORTACAVAL SHUNTS

This paper is based upon experience gained in the establishment of portacaval shunts in ten cases (five splenorenal anastomoses, and five portal vein to vena cava anastomoses). As in the case with points on technic, the experience is too limited to express more than formative opinions regarding the selection of cases for operation and the type of shunt indicated in the individual case.

It goes without saying that the selection of cases is based upon a careful history, physical examination and special studies, including kidney function studies. Convincing clinical evidence of portal hypertension should be procurable in the vast majority of cases preoperatively. Furthermore, one can accurately predict, on the basis of liver function chemistry, whether the

portal hypertension is due to intrahepatic (portal cirrhosis) or extrahepatic portal bed block.

A case of splenomegaly giving a history of hematemesis or gastro-intestinal bleeding in association with the presence of anemia, leukopenia, thrombocytopenia and a normal liver function chemistry may be safely diagnosed as congestive splenomegaly due to extrahepatic portal bed block. The splenomegaly may be discovered in infancy or childhood to suggest the presence

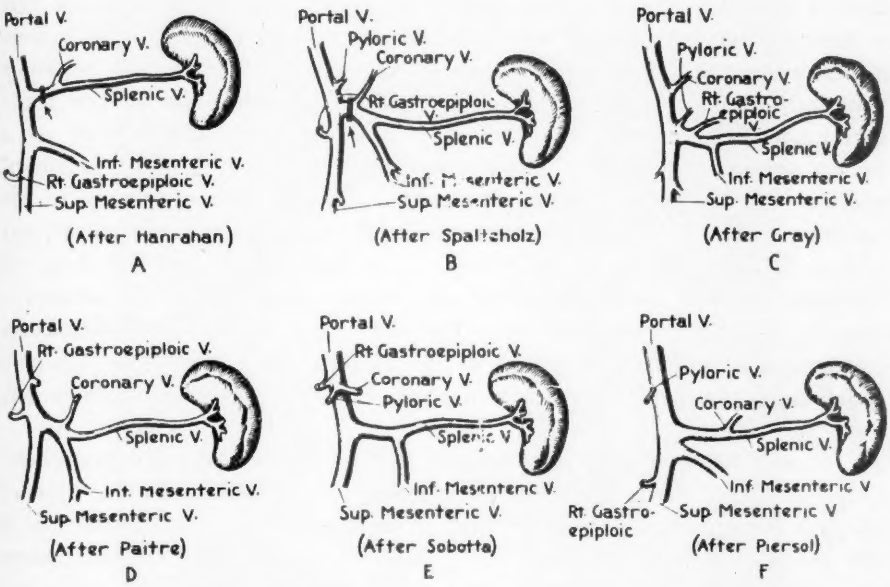

FIG. 4.—Illustrating the anatomic variations in the major branches of the portal vein. Note the effect of a splenic vein block proximal to the origin of the coronary vein.

of portal vein atresia, atresia of the splenic vein at its origin; or, in other cases to follow the history of an injury suggesting splenic vein thrombosis. Rousselot[9] has presented evidence to suggest that congestive splenomegaly when due to a block in the splenic vein may or may not be accompanied by a vicious hypertension localized to the branches of the coronary vein of the stomach (which anastomose with the esophageal veins) in accordance with whether the coronary vein arises from the splenic or the portal veins. If the coronary vein happens to arise from the portal vein, thrombosis of the splenic vein would not, of course, be expected to cause esophageal varices. Or the same would hold in cases in which the coronary vein arose from the splenic vein, provided the thrombosis of the splenic vein were limited to a region of the splenic vein distal to the entrance of the coronary (see Fig. 4). Since four out of the six anatomists list the coronary vein as normally arising from the splenic vein, the chances of a vicious hypertension involving the coronary vein system of the stomach as a threat to hemorrhage from esophageal varices is a real one.

In view of the above facts splenectomy alone as a treatment for congestive splenomegaly should be limited to those cases of splenic vein thrombosis in which the coronary vein arises from the portal vein or, if arising from the splenic vein, the obstruction in the splenic vein must be distal to its origin. Figure 5 illustrates a case in which the above indications were not observed, unfortunately, and there are more about the country. This venogram, made at operation following the injection of a branch of the coronary vein with 35 per cent diodrast, shows the course of the coronary vein (note arrows) directly downward toward the splenic vein. The fact that this patient had a massive hemorrhage six months following the removal of the spleen indicates that the splenic vein was blocked between the origin of the coronary vein and the junction of the splenic with the portal vein. Manometric readings made on a branch of the superior mesenteric vein and another on a branch of the coronary vein at the time of splenectomy had shown a normal reading for the superior mesenteric and an elevated venous pressure in the coronary vein. This finding corroborates the venogram and the subsequent clinical behavior following splenectomy. If the splenectomy had been, at the time, followed by a splenorenal anastomosis, in this case further hematemesis may have been avoided. The only hope of the postsplenectomy bleeders of this particular group would seem to be a vein graft bridging anastomosis of the coronary vein to the left renal vein *via* a thoraco-abdominal, lesser sac approach.

In cases of congestive splenomegaly with a normal liver function chemistry, indicating the presence of extrahepatic portal bed block, the type of surgical therapy must be determined at operation. A quick inspection of the portal vein will determine the presence or absence of cavernomatous transformation. Atresia of the portal vein at the portal fissure may be less easy to recognize. Obstruction sites in the splenic vein, on the other hand, are often extremely difficult to palpate or demonstrate. Venous pressure readings are essential. Figure 6 illustrates a simple device for obtaining readings. Blood pressure in the portal radicals varies normally from 80 to 100 mm. of water. A reading above 110 mm. of water should definitely be considered above normal in our experience. At the outset, a pressure reading should be taken from a branch of the superior mesenteric vein; if this is elevated, it may be taken as evidence of a block in the superior mesenteric vein, portal vein or intrahepatic portal block. A normal reading from a branch of the superior mesenteric vein and an elevated reading from a branch of the coronary vein of the stomach would indicate a block in the splenic vein, and, furthermore, strongly suggest that the coronary vein originates from the splenic vein distal to the site of obstruction. This evidence alone would make us favor performing a splenectomy followed by a splenorenal anastomosis rather than a splenectomy alone. In a case of congestive splenomegaly in which the superior mesenteric pressure is normal, the splenic vein pressure elevated but the coronary vein pressure approximately normal, we would be inclined to perform a splenectomy only. Venography following

the injection of 15–20 cc. of 35 per cent diodrast in a branch of the coronary vein is useful in confirming the site of origin of the coronary vein.

In most cases of cavernomatous transformation of the portal vein splenorenal anastomosis is likely to be the only type of portacaval shunt it is practical to use. In some cases of cavernomatous transformation, however, or

Fig. 5.—Venogram following diodrast injection of a branch of the coronary vein. Arrows point to coronary vein descending toward the splenic vein.

cases of atresia of the portal vein at the portal fissure, in which the spleen has been previously removed, it may be possible to do a portal to vena cava anastomosis using a vein graft. We found it to be feasible in one case.

We are coming to believe that anastomosis of the portal vein to the vena

cava may be preferable to splenorenal anastomosis for the treatment of Laennec's cirrhosis of the liver, though it will take time and more experience to settle this point. In cases of cirrhosis of the liver, having very large spleens, it is logical and safe to ligate the splenic artery in addition if, after the portal vein is implanted into the vena cava, the spleen should fail to shrink satisfactorily at the time. We have not found it necessary to do this so far.

Fig. 6.—Photograph of a manometer and tubing arrangement suitable for taking portal pressures and the injection of diodrast for making venograms of the portal bed.

It is our feeling that the larger volume of blood shunted by the portal vein when implanted in the vena cava in comparison to the amount handled by the smaller splenic vein in a splenorenal anastomosis may more effectively lower the portal tension and reduce the tendency to ascites. In regard to the latter, however, the possible use of the left ureter following nephrectomy to drain off ascitic fluid when properly implanted in the peritoneal cavity must not be lost sight of. Studies are in progress in this direction.

Discussion: There are two important hemodynamic factors that are known to affect the immediate success or continued patency of blood vessel anastomoses, namely, (1) intravascular pressure; and (2) rate of blood flow. Surgeons experienced in the suture anastomosis of blood vessels are about as certain of the success of arterial anastomoses as they are certain of the failure of vein anastomoses. Arteriovenous anastomoses, on the other hand, can be counted upon to remain patent indefinitely and with unfailing regularity. In the former the high intravascular pressure is an important factor favorable to success. In the latter (arteriovenous anastomosis), in addition to the favorable factor of pressure there is an extreme high rate of blood flow, a result of shunting blood from a high pressure (arterial) system to a low pressure (venous) system. These factors insure the perpetuation of anastomoses even though they be formed by the passage of bullets, knives, *etc.*

It seems likely that the important reason for failure of carefully performed suture anastomoses of veins is the low intravascular pressure. The normal systemic vein pressure is under eight millimeters of mercury. This unfavor-

able factor in combination with cessation of flow due to vasospasm may initiate clotting along the suture line which often rapidly propagates to complete occlusion of the vessel at the site of the anastomosis. The above facts being true regarding the anastomosis of veins in general, the question may very properly be asked, what chance is there of a portacaval shunt remaining permanently open? In the first place, a portacaval anastomosis done for the relief of portal hypertension has the favorable factor of dealing with a greatly increased venous pressure. The portal pressures in our cases ranged from 260 mm. of water (20 mm. Hg.) to over 500 mm. of water (40 mm. Hg.).

A second factor important in perpetuating the patency of portacaval shunts is the extremely high rate of blood flow—the identical factor so important in maintaining the persistence of traumatic arteriovenous fistulae. Though the pressure differential be not of the same magnitude in comparing the two types of shunts, the principle is the same, namely, the shunting of blood from a high pressure (portal) circuit to a low pressure (vena cava), low resistance to flow circuit. It is not unreasonable to venture the statement that the rate of blood flow through a good-sized portacaval shunt may far exceed that of a large artery. In view of the above hemodynamic facts favorable to the perpetuation of portacaval shunts, success or failure in the individual case would seem, then, to depend upon technical factors.

There is full agreement that the ideal technic in blood vessel anastomosis embraces intima-to-intima coaptation without the interposition of a foreign body in contact with the flowing blood. It is conceded that suture anastomoses, when done with meticulous care, may closely approximate the above ideal. But, in our opinion, its application to the establishment of portacaval shunts is impractical. This is because of the presence of complicating factors that are likely to compromise, at some point, the execution of a uniform technic so essential to the success of vein anastomosis, namely, intima-to-intima coaptation without foreign body contact with the flowing blood. For example, it must be remembered that a beautifully performed suture anastomosis, up to a point, may suddenly be ruined by the malplacement of two or three stitches, the execution of which had been compromised by unfavorable circumstances, such, for example, as inadequate exposure, *etc.*

It is our opinion that the nonsuture method of blood vessel anastomosis is peculiarly suited to the establishment of portacaval shunts. The method embraces the ideal feature of intima-to-intima coaptation without intervening foreign body (suture). It is important to remember that the above feature is approximated only in the "perfect" suture anastomosis whereas, it is automatically assured at the completion of the nonsuture anastomosis, irrespective of the difficulties of exposure, *etc.*, that may be encountered during its performance.

In addition to the greater technical ease with which portacaval shunts may be established, using the nonsuture method, it requires less time than

the suture technic. Time is particularly an important factor in the performance of a portal vein to vena cava anastomosis (Eck fistulae). This is because it is necessary to completely occlude the vena cava proximal to the entrance of the renal veins during the period the vena cava is actually open, *i.e.,* while doing the anastomosis. Serious kidney damage may result from prolonged blockage of the renal veins. It seems likely that this may have been the causal factor in some of the fatalities reported in the early literature following the performance of Eck fistulae employing the suture technic. One may cut down this period of occlusion of the vena cava to a minimum using the nonsuture technic—the time elapsed from the time of incision of the vena cava to completion of the anastomosis may be, and should be, as little as ten minutes.

In conclusion, we may state that every one of the ten cases of portacaval shunts (five splenorenal and five Eck fistulae) went through a successful postoperative convalescence. The interval following operation has been too short in some to judge the results. However, in six of the ten cases the improvement has already been so outstanding as to justify continuing the procedure. The Eck fistula operation is better tolerated by the patient, probably because of less blood loss during the procedure.

SUMMARY

A nonsuture method of establishing portacaval shunts by anastomosing the splenic vein to the renal vein or the portal vein to the vena cava, employing vitallium tubes is described and illustrated. The indications for the employment of the two types of shunts are discussed and the technic for each described.

The hemodynamics of portacaval shunts are reviewed with special reference to those features of the nonsuture technic that favor maintained patency of the anastomoses.

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THE USE OF VITALLIUM TUBES IN STRICTURES AND ABSENCE OF THE COMMON BILE DUCT*

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STRICTURES OF THE BILE DUCTS are extremely serious lesions since they are so commonly secondary to operative procedures upon the biliary tract, and because operative correction has up to date been relatively unsatisfactory. They must be classified into various types since some are extremely serious and others readily correctable. For example, a short stricture of the common duct which has sufficient proximal and distal duct left for approximation can be reanastomosed with little difficulty and with a favorable outcome. Much more serious is the total absence of the common duct which unfortunately is more common. Undoubtedly portions of the ducts, particularly the distal end, may remain buried in adhesions, thus, making it very difficult to find. The proximal end is usually readily found because of the back pressure of bile which will usually create a bulging structure. It is the group in which no common duct can be found which up to date has yielded such poor results.

In our series of 23 cases of stricture or absence (including two carcinomas of the common hepatic duct) encountered during the past six years, no duct could be found on ten occasions except the stump at the hilus of the liver. In this group we adopted the use of the vitallium tube as introduced by Pearse.[1] After some preliminary trials with different types of procedures, we believe we have established a few principles which have helped us in arriving at a method which yields at least fairly good results in this complete defect which is so difficult to correct.

ETIOLOGY OF STRICTURES OF THE COMMON DUCT

The causes of benign strictures of the common duct can be divided into five major groups, as shown in Table I. As stated, we conducted a study of 23 consecutive cases encountered during the past six years. Eliminating two which were caused by a malignant tumor of the duct itself, 48 per cent were so definitely related to the original operation of cholecystectomy that trauma appears to have been the direct cause of the stricture. In another 28 per cent the relationship of the stricture to the operation was probable, thus, suggesting that 76 per cent were related to the operation. This is slightly less than the figure of 80 per cent given by Cattell,[2] and of 90 per cent by Walters.[3]

In 9 per cent of our cases the appearance of symptoms before the original

* This article was to have been presented before the Annual Meeting of the American Surgical Association, May, 1945.

operation made it appear that the stricture was definitely of the inflammatory type.

In the inflammatory group, ulceration produced by stones may be an important factor in etiology, but other factors including cholangitis and pylephlebitis appear to be more specifically related to it. When the stricture develops slowly after an operation from which the patient recovered slowly because of fever and purulent drainage, there is strong probability that an abscess adjacent to the duct was the primary factor in its destruction. In the entire series (excluding carcinoma) there were six (28 per cent) which we classified as being of the inflammatory type.

We encountered three cases of stricture of the terminal end of the common duct secondary to pancreatitis, constituting an incidence of 14 per cent. The pancreatitis in these three cases was of the severe type from the pathologic standpoint, insofar, as destruction of the pancreas was quite far advanced, particularly in two cases. As would be expected, the strictures were located in the terminal end of the duct and confined chiefly to that portion of the duct passing through the diseased pancreas. From the standpoint of discussion in the literature, these strictures are apparently quite uncommon. They will be discussed in detail in another publication.

Table I

CAUSES OF BENIGN STRICTURES OF COMMON BILE DUCT

1. Operative trauma:
    - (a) Excision, ligation or incision
    - (b) Clamped while controlling hemorrhage
    - (c) Cystic duct ligature too close
    - (d) During gastrectomy
    - (e) Following choledochostomy (rare)
2. Ulceration due to gallstones
3. Inflammation
    - (a) Related to cholangitis
    - (b) Abscess about duct
    - (c) Pylephlebitis
4. Secondary to pancreatitis
5. Tumors and multiple cysts

PREVENTION OF STRICTURES

The seriousness of strictures of the common duct makes it imperative that we do everything possible in their prevention. On many occasions, as will be discussed later, ulceration due to gallstones appears to be a reasonably good explanation. Therefore, *early removal of stones* would in reality be indicated. Opinions differ as to whether stones should be removed when no symptoms are being produced by them. Some surgeons are of the opinion that all gallstones should be removed. In elderly people who are asymptomatic, the chances of damage being inflicted from gallstones appears so slight to the authors that cholecystectomy is scarcely indicated. However, we are definitely of the opinion that stones should be removed along with the gallbladder in young patients even though asymptomatic. The chief problem in our minds from that standpoint is the age-border. In general, we feel that if asymptomatic stones are found in people below the age of 40

to 45 and their health is sufficiently good to predict normal life expectancy, the stones should be removed along with the gallbladder.

To eliminate the possibility of strictures resulting from an operation, certain principles in the *prevention of operative trauma* must be adopted and adhered to closely. The authors believe it makes very little difference whether the surgeon starts his dissection from the fundus or from the cystic duct end when performing a cholecystectomy, although they usually start the dissection from the cystic duct end. If adhesions are so dense that dissection at this point becomes dangerous, it seems obvious that the plan should be reversed and dissection started from the fundus, which will allow better isolation of the cystic duct although more hemorrhage will be encountered particularly from the liver bed. Unquestionably this change in technic will minimize the possible damage to the common duct when visualization is so difficult. Safety in gallbladder surgery is strongly dependent upon *good exposure*. The wound should be enlarged until good exposure is obtained. The *cystic duct should be dissected* out down to the common duct until the latter structure can be exposed. The duct and artery should be *tied separately* because ligation in one mass is so inaccurate that the common duct could easily be pinched by the ligature. When working in the region of the common duct, the surgeon must abide by the rule *not to cut any structure until it is completely identified.* He should always dissect by direct vision and not rely on anatomy, although a knowledge of anatomy is of course entirely essential to any surgeon. The reason why the surgeon must not trust the rules of anatomy in dissection about the common duct is because anomalies are so frequent in this area. The *operator must not hurry* while dissecting in the region of the cystic and common duct.

Although strictures very seldom result from the simple procedure of *choledochostomy, this operation should be undertaken with extreme* care. Obviously the incision in the common duct for the extraction of stones should be in the longitudinal direction. Particular care must be taken lest the duct be torn transversely. If a transverse tear is produced, the stage is probably set for the development of a stricture later unless an extremely careful repair is made.

METHODS OF REPAIR

The method of repair depends entirely upon the type of defect encountered. Defects can be divided into four major groups from the standpoint of the type of technic indicated, as will be discussed later.

There are certain principles in treatment which apply to all types of repair. It is highly desirable to approximate mucosa to mucosa particularly if a supporting tube is not used. Obviously, one should attempt to save all the duct possible. However, in the dissection of the ducts one should minimize the mobilization so as to preserve vascular supply. Nevertheless, on certain occasions when the distal stump is present but short, the duodenum will have to be mobilized to allow contact with the proximal stump. Any tube

must be anchored securely, although no suture will hold longer than a few weeks. Even though silk or wire sutures are applied, they will cut through in due time and the suture fixation will be eliminated. Utilization of rubber tubes has been tried by numerous surgeons but in general the results have not been satisfactory largely because they are usually passed within a short time; within 3 to 12 months after passage of the tube signs of obstruction usually develop. However, we do have two or three patients who have sustained fairly good results following suture over a rubber tube. If they are retained there is a slightly greater tendency for precipitation of bile salts in the lumen than in the lumen of vitallium tubes. It appears well proven that rubber itself acts strongly as a foreign body. Tissue appears to make all effort to extrude the rubber tube. On the contrary, from data up to date, it appears that vitallium is well tolerated by tissue and does not act as a foreign body. All of the rubber tubes which we have inserted (about 15) have been passed. Of 14 vitallium tubes which we have inserted within the last few years, three have been passed.

Although most of the discussion in this presentation is related to the use of vitallium tubes in strictures or absence of the common duct, we wish to emphasize that wherever possible anastomosis should be made with no more than temporary intraluminal support. The indications which we have arrived at will be discussed later under the different types of defects. As suggested above, effort should be made to bring the two ends of the ducts together so that they can be approximated with sutures. However, if this approximation can be achieved only by tension, it will probably not hold. In general, the application of a T-tube to bridge defects achieves the purpose of function quite well. The chief disadvantage of a T-tube is that when wearing it permanently the rubber tube acts as a source of mental irritation to the patient and frequently a source of infection. Almost invariably stricture will result within a variable time after removal of a T-tube if it is inserted in the anastomotic line. Insertion of the tube 2 to 3 cm. distal to the anastomotic line, as performed by Cattell,[2] minimizes stricture formation. Results following the insertion of a T-tube to bridge a defect are commonly so satisfactory that many surgeons suggest this type of operative repair for stricture of the common duct, leaving the tube in permanently.

1. *Local Stricture of the Common Duct.*—This type of lesion is the most readily repaired and, in general, will give the most satisfactory results. Unfortunately it is relatively uncommon. In 23 cases of stricture of the common duct repaired by various technics we encountered a local stricture in only one case (4.5 per cent). In a series of 80 cases reported by Walters,[3] such strictures involving no more than 1 to 2 cm. of the duct were encountered in 15 per cent. In the opinion of the authors there is not a very strong indication for insertion of a vitallium tube in local strictures, although we utilized the vitallium tube in the case just mentioned with very good results. As a matter of fact, cases originally reported by Pearse,[1] who introduced the use of the vitallium tube in repair of bile duct defects, were examples of

this type, *i.e.,* local stricture. It is possible that anastomosis of two ends of the duct together over an arm of a rubber T-tube inserted 2 to 3 cm. distal to the anastomotic line, as practiced by Cattell and others, may be just as satisfactory in such cases, but would not be applicable to the more common defect in which no common duct whatever can be found.

If a vitallium tube is used, the type with a flange on the shaft but without the funnel-shaped end, would appear desirable. The flange on the shaft should prevent the tube from slipping down into the common duct if a

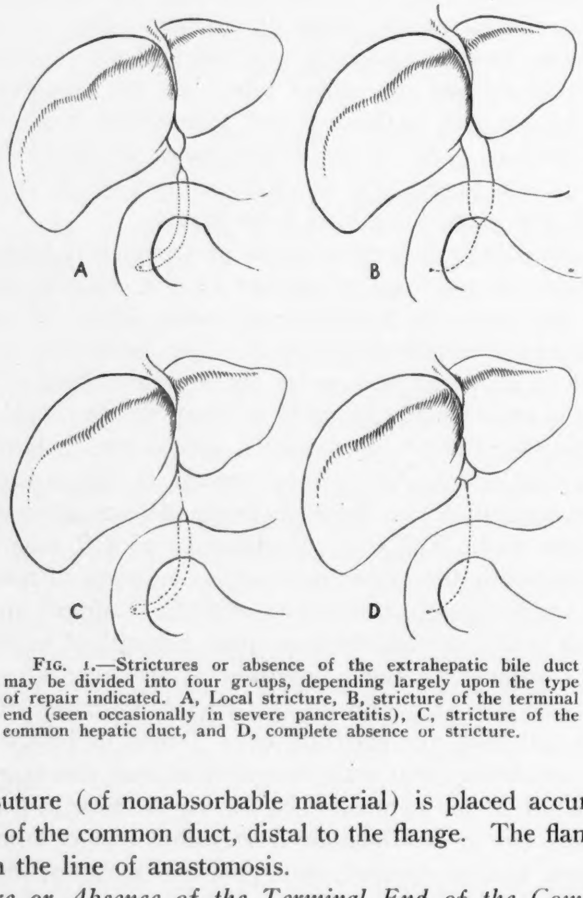

FIG. 1.—Strictures or absence of the extrahepatic bile duct may be divided into four groups, depending largely upon the type of repair indicated. A, Local stricture, B, stricture of the terminal end (seen occasionally in severe pancreatitis), C, stricture of the common hepatic duct, and D, complete absence or stricture.

purse-string suture (of nonabsorbable material) is placed accurately around the distal end of the common duct, distal to the flange. The flange is allowed to protrude in the line of anastomosis.

2. *Stricture or Absence of the Terminal End of the Common Duct.*—This type of lesion will likewise be uncommon but is quite readily repaired. If a few centimeters of the common hepatic duct are present, the authors are definitely of the opinion that transplantation of the duct into the duodenum should be the first operation tried. There is a great tendency for this type of repair to result in a stricture, unless careful efforts are made to attach *mucosa to mucosa* in this repair. If desired this anastomosis may be performed around a rubber tube, several centimeters in length, which protrudes into the lumen of the duodenum. This tube will remain several days, or

perhaps longer, but will maintain patency for passage of bile during the early postoperative period when edema might otherwise block the duct. This is the type of repair so commonly performed now during resection of the head of the pancreas for carcinoma. Several surgeons[1, 4, 5] have complained of development of cholangitis, with chills and fever, following this

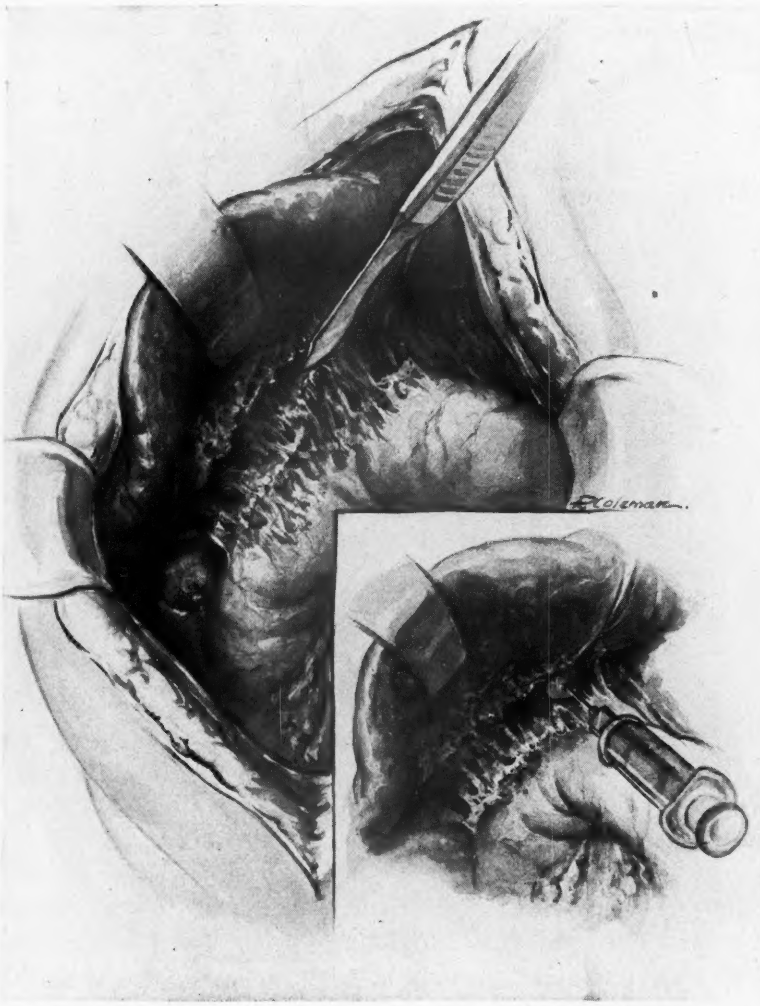

FIG. 2.—Exposure through the dense adhesions is best achieved by dissecting between the liver and intestines with the knife or dissecting scissors, starting from the lateral side. If the duodenum has been attached to the stump of the duct at the hilus previously, the bile duct will be encountered and opened before the portal vein or hepatic artery is reached. Insert shows aspiration of hilus region to identify the duct stump from other structures.

type of repair, although in the majority of cases no symptoms will be encountered at least for several months or a year or two. If a stricture forms or chills become frequent, some type of anastomosis to an isolated arm of the jejunum, as described later, may be advisable.

3. *Absence or Stricture of the Common Hepatic Duct.*—Lesions of this type are more difficult to repair than the two previously described, largely because anastomosis of the duct at the hilus of the liver cannot be achieved with ideal technic. The authors are of the opinion that insertion of a vitallium tube with the funnel end projecting into the stump of the common hepatic

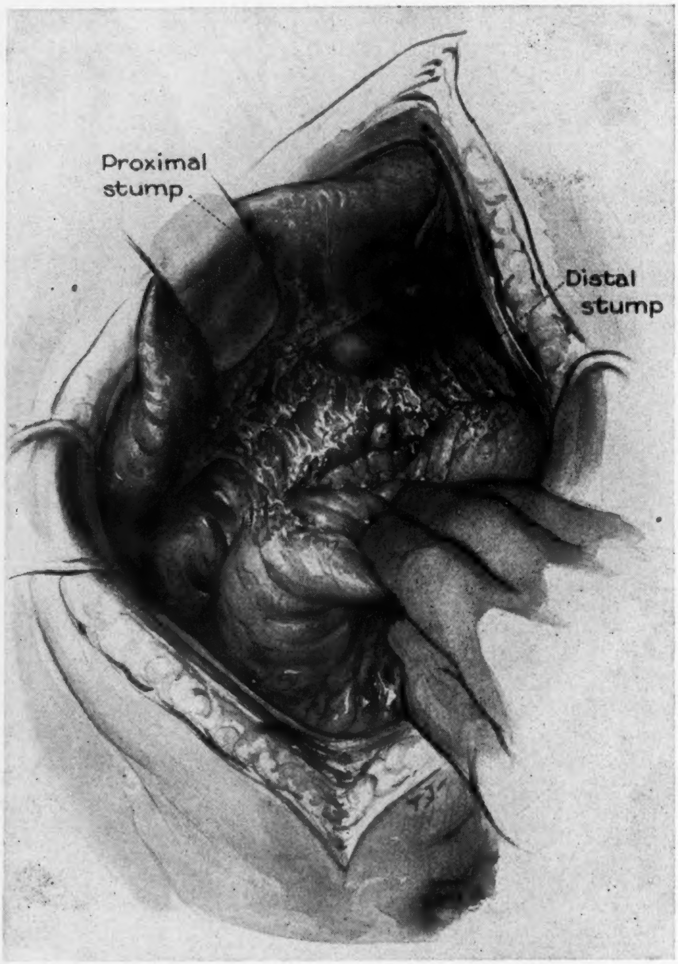

FIG. 3.—Attempt should always be made to find the distal end of the common duct because of the great value of the sphincter of Oddi. Incision of the peritoneum on the lateral and superior side of the duodenum will aid in the search.

duct at the hilus and the lower end protruding into the common duct, as first performed by Clute[6] in this type of defect, is the procedure of choice. Application of a purse-string suture around the opening of the stump of the duct at the hilus serves to anchor the tube particularly when a tube with the funnel-shaped enlargement at one end is used. The distal end of the common duct is then brought up and the tube inserted into it. If possible

the duodenum should be mobilized so that the end of the common duct can be sutured to the hilus of the liver, with the flange in the shaft protruding. If the terminal end of the duct cannot be sutured to the hilus, it *should be anchored firmly* with several interrupted sutures to adjacent tissue because of the danger of the duct slipping off the tube.

4. *Absence of the Common and Common Hepatic Duct.*—When no remnants of the external duct can be found the problem of repair becomes much more difficult. Obviously the only possible method of correction would be

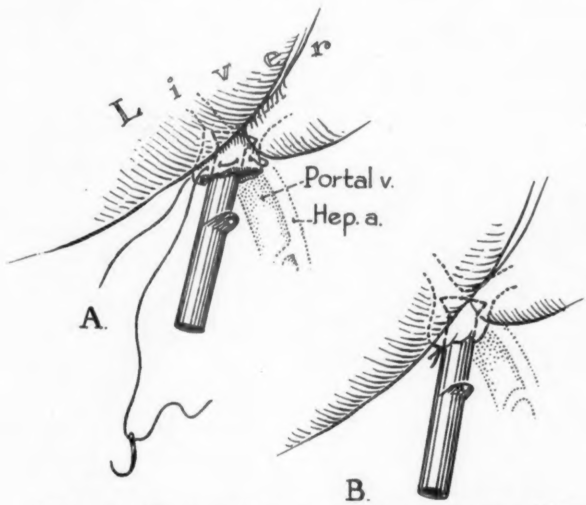

FIG. 4.—A. The stump of the duct is incised or dilated sufficiently to allow insertion of the "funnel" end of the vitallium tube. A purse-string suture is applied,—preferably before the tube is inserted. B. A water tight connection is achieved by tying the suture. If the bifurcation of the ducts is located close to the surface the tube shown may not allow free drainage; a tube with a forked or Y-shaped end will be preferable, since both ducts can then be cannulated.

to anastomose the stump of the common hepatic duct at the hilus of the liver to a loop of intestine. The conventional method utilized up until recent years has been to perform this anastomosis between the hilus stump and the duodenum, usually over a short rubber tube which remains in position for a variable length of time but which will invariably be passed on, since it protrudes into the duodenum where food will dislodge it and carry it along. Dragstedt, and associates,[7] have reported the successful use of a modification of the conventional method of repair in a patient with complete absence of the duct; they constructed a tube from the wall of the duodenum and attached it to the stump of the hepatic duct at the hilus.

Inability to obtain a good anastomosis, and the lack of an appreciable amount of duct wall are largely responsible for the poor results in plastic operations when no duct can be found. A stricture forms in the line of anastomosis in a majority of the cases, although now and then the patient has a good result with very little if any evidence of obstruction or cholangitis. Cholangitis is the pathologic lesion feared in any repair of this type (*i.e.,* total

absence of the duct). Symptoms consist primarily of chills and fever; however, the great danger is development of multiple abscesses of the liver.

As will be discussed later under "Comment," we are of the opinion that in addition to stricture formation, reflux of food and intestinal secretions into the intrahepatic ducts is very important in the development of cholangitis. If this is true we should obviously adopt an operative procedure which would minimize that complication. Since we have no method of constructing an artificial sphincter it appears we should attempt mechanically to prevent reflux of

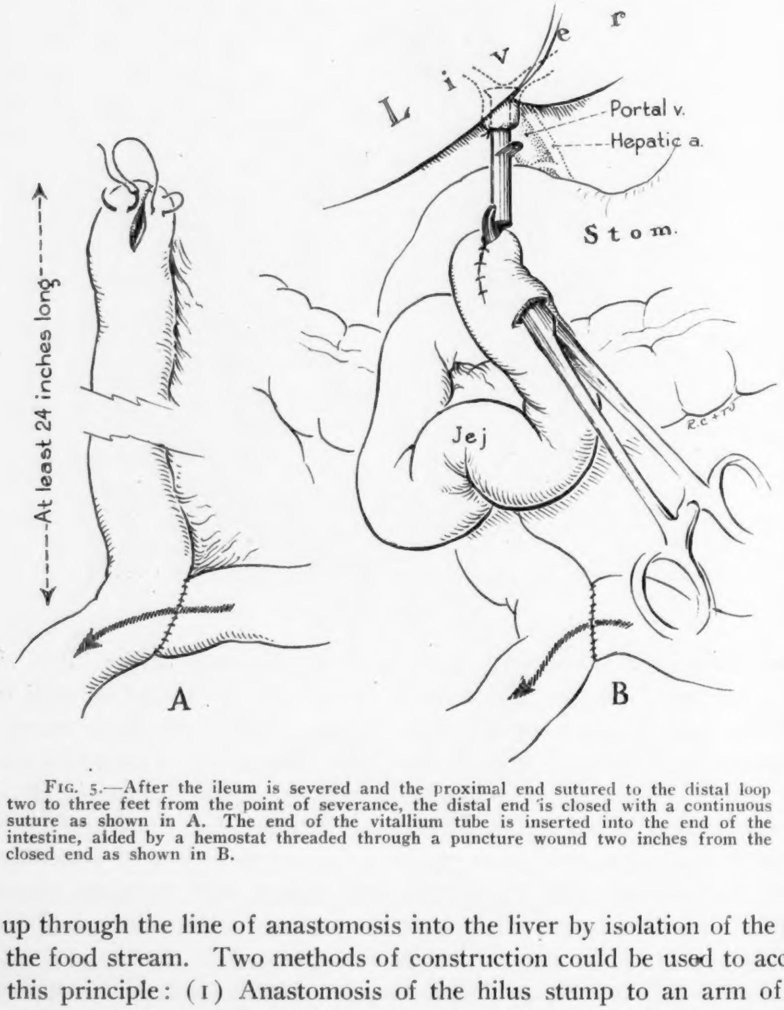

FIG. 5.—After the ileum is severed and the proximal end sutured to the distal loop two to three feet from the point of severance, the distal end is closed with a continuous suture as shown in A. The end of the vitallium tube is inserted into the end of the intestine, aided by a hemostat threaded through a puncture wound two inches from the closed end as shown in B.

food up through the line of anastomosis into the liver by isolation of the area from the food stream. Two methods of construction could be used to accomplish this principle: (1) Anastomosis of the hilus stump to an arm of the jejunum after the Roux principle (see Figs. 5 and 6); or (2) attachment of the hilus to a loop of jejunum in which an anastomosis is performed between the two loops at a distance from the hilus anastomosis (see Fig. 7). We have utilized both of these principles in construction of an intestinal loop to transport the bile, but have had better results with the method utilizing the single arm

of jejunum utilizing the Roux principle. We will, therefore, describe this method in more detail.

(1) *Anastomosis of the Hilus Stump to a Single Arm of Jejunum*

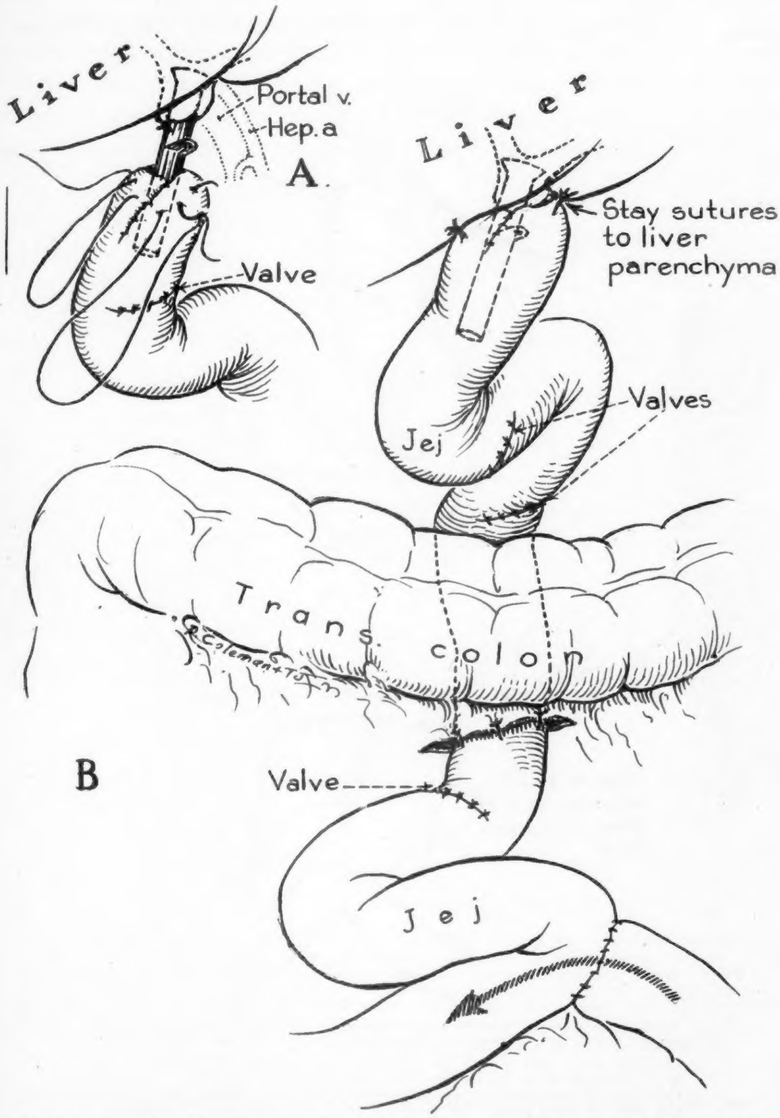

FIG. 6.—A. The connection between intestine and distal end of the vitallium tube is made tighter by application of a purse string suture of silk or cotton which is more easily applied before the tube is inserted. B. The end of the intestine is anchored against the liver by interrupted sutures all of which should be inserted before any one is tied.

*Utilizing the Roux Principle.**—Commonly the patient has already had two

* Since preparation of this manuscript Allen has published an article (ANNALS OF SURGERY, 121, 412, 1945) describing a similar use of the Roux arm of jejunum, except that a rubber tube was used.

or three operations before coming in for repair of the stricture. Adhesions are practically always extremely dense because of the infection and irritation of the bile incident to the biliary fistula. Therefore, care must be exercised in dissecting down toward the region of the common duct lest the intestine be damaged. The colon is usually densely adherent to the abdominal wall

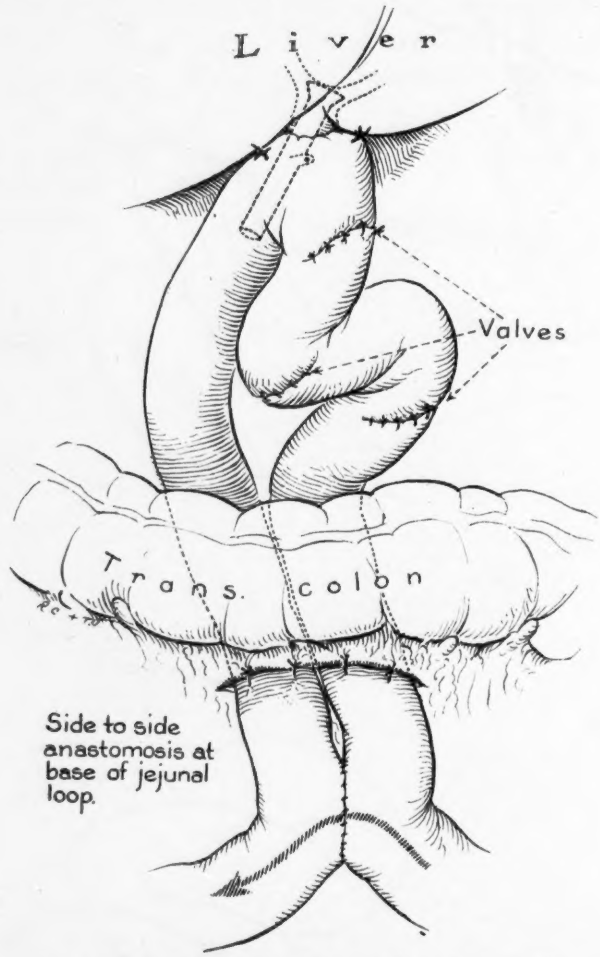

FIG. 7.—An alternative and shorter method of constructing an outlet of bile to a defunctionalized portion of intestine is to insert the vitallium tube into a loop of jejunum. An entero-enterostomy should be performed 18 to 24 inches from the point of insertion of the tube. However, in our experience this method has ultimately been followed (as late as two years) by severe cholangitis presumably because of reflux of food and intestinal contents into the liver. Construction of valves as illustrated in Figure 8 might minimize this tendency.

and ventral surface of the liver. The duodenum is usually plastered densely against the hilus of the liver beneath the colon. Dissection, bluntly or by scalpel, can best be performed by staying close to the ventral surface of the liver and working from the lateral side down towards the region of the common duct. Effort should first be made to locate the common duct or its

remnants. The duodenum can be mobilized by incising the peritoneum along its right border hoping to find a remnant of the distal end of the common duct as the duodenum is rotated toward the midline. However, the dense adhesions which are so consistently present make isolation and discovery of the distal end of the common duct extremely difficult and, in fact, unlikely. As will be discussed later, in the majority of cases in our series we were unable to find any remnant of the common duct except indefinite fibrous bands. An incision can be made in the duodenum over the sphincter of Oddi hoping

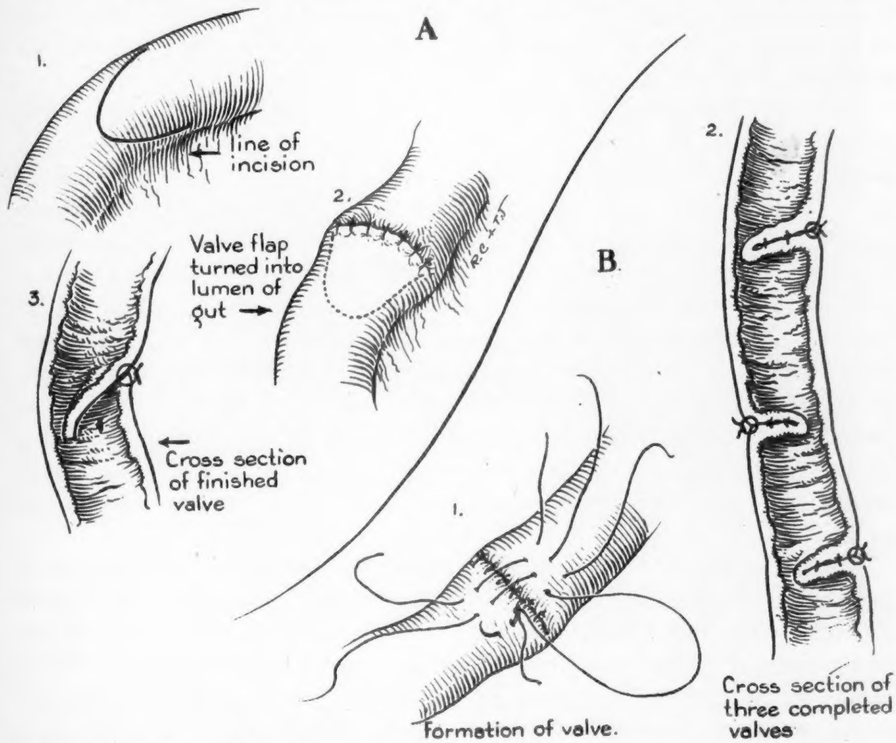

FIG. 8.—In our experience the construction of valves of one of the two types illustrated above eliminates reflux of food (as indicated by barium studies) and has minimized or eliminated the cholangitis. A. A valve may be constructed by outlining a flap of intestine and closing the defect after inverting the flap into the lumen. B. The creation of baffles by a double layer of sutures infolding the wall of the intestine into the lumen are probably as effective as A and are much more simply constructed. At least three of these folds should be made. No claim for originality is made for these valves since similar ones have been reported for various purposes during the past several decades.

to find this structure and, thus, locate the distal end of the duct. We have been extremely unfortunate, however, in locating the duct or sphincter by this method. We have recently discovered an explanation of this difficulty, insofar, as we have found a stricture at the sphincter level on several cases, as will be described in another publication. If an incision is made in the duodenum it should be made longitudinally and closed transversely, after the Mikulicz technic.

If it is demonstrated that no common duct is available, attention should be centered on the localization of the stump of the hepatic duct at the hilus.

If another plastic operation, such as hepatoduodenostomy, has been performed previously, the anastomotic line can be located easily by incising from the lateral side toward the point of fixation between the hilus and the duodenum. This will cut into the strictured area without endangering the portal vein or hepatic artery. The safety of this approach to the hilar structures is appreciated more fully when we realize the seriousness of incision into the portal vein or hepatic artery. If no previous operation of this type has been performed, the stump of the duct is located by aspiration with an hypodermic syringe and needle. On some occasions the duct appears actually to be buried in the liver tissue. Frequently it contains white bile and not normal colored bile.

The lack of sufficient amount of hepatic duct for an anastomosis is ample explanation for the high percentage of failures without the use of a permanent tube of some type. Insertion of a vitallium tube through the stump of the hepatic duct into the lumen of a functioning intestine (usually duodenum) would appear to result always in passage of the tube sooner or later. We have not attempted this type of anastomosis because it appears to us that the food stream would inevitably dislodge the tube. Certainly it has been true with the use of rubber tubes. As indicated above, we have utilized the principle of using a blind loop of jejunum to anastomose to the stump of the hepatic duct. We have likewise discovered that the amount of intestine intervening between the anastomotic line at the hilus and the intestinal anastomosis where the food enters must be at least 24 inches (see Fig. 6) because food can regurgitate for several inches into a blind defunctionalized loop.

After the stump of the common hepatic duct is located at the hilus the jejunum is identified and severed about one foot or more from the ligament of Treitz. The distal end is closed by inverting with a single suture of continuous catgut. The end of the proximal loop is then anastomosed to the distal loop at least two feet from the end which is to be attached to the duct at the hilus of the liver. It is immaterial which type of anastomosis is utilized, although the end-to-side seems appropriate to us; if desired, the end of the proximal loop could be closed and a side-to-side anastomosis performed. The arm of the jejunum to be anastomosed to the stump of the duct is then placed in position to see if the mesentery of the jejunum is long enough to reach around the colon and allow anastomosis between the duct and intestine without tension. If the mesentery is long enough an anterior anastomosis is performed. If it appears too short to reach around the colon then it is preferable to make an opening in the mesocolon and draw the arm of the jejunum up through this opening. If this is done, the edges of the opening in the mesocolon must be anchored to the jejunum and its mesentery, lest loops of intestine invaginate into the opening in the mesocolon and become obstructed. The opening of the common hepatic duct at the hilus is then dilated to a size which will allow introduction of the funnel end of the vitallium tube. With a probe or curved blunt instrument the right and left

hepatic duct can be located easily. Occasionally the duct will divide so close to the hilus that the ordinary tube will be blocked by the septum between the ducts. Under such circumstances, a tube with a "Y" end should be used so that each duct is cannulated. It so happens that in all except one of our cases the duct has divided high enough in the liver to allow us to use the ordinary vitallium tube with a funnel end. A purse-string suture of silk or

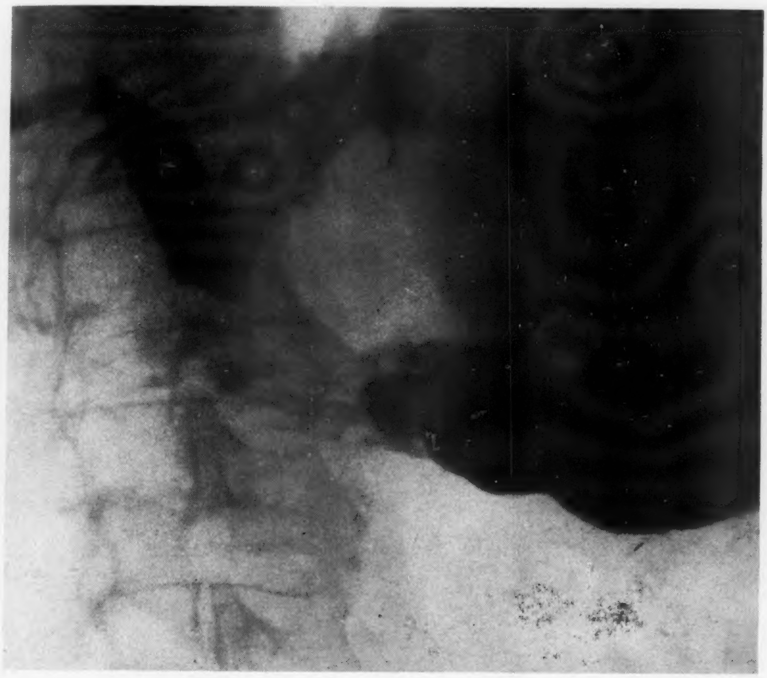

FIG. 9.—X-ray of the liver after a barium meal in a patient who had had the conventional anastomosis between the duodenum and stump of the common hepatic duct for a complete absence of the common duct. The anastomosis was performed over a rubber tube which was passed a few weeks later. Note the massive regurgitation of barium into the dilated intrahepatic ducts. He is having frequent chills and needs another type of repair.

cotton is then placed around the end of the duct and the tube inserted. Tying the suture should anchor the tube securely. Since the portal vein lies adjacent to the bile duct, there is danger of puncturing it when applying the suture on this side. If there is doubt about the position of the portal vein when the purse-string suture is applied on the medial side, this area may be aspirated with an hypodermic needle. If at least 2 or 3 mm. of tissue intervene between the duct and the vein, the suture can be taken without endangering the portal vein. It would be highly undesirable to place the suture through the wall of the vein since the trauma might encourage thrombosis or thrombophlebitis. A purse-string suture is then applied around the center of the line of closure at the end of the arm of jejunum. A small opening is made about two inches from the end and a curved Pean forcep inserted (see Fig. 5B). A tip of the forcep is threaded through the closed end of

the jejunum between the linear sutures and through the purse-string. The end of the vitallium tube is then grasped and pulled into the end of the jejunum and the purse-string suture tied. This then allows the intestine to be fixed against the hilus of the liver with the certainty that the tube is adequately threaded through the end of the jejunum into its lumen. Interrupted sutures are placed anchoring the end of the intestine against

FIG. 10A.—X-ray of liver after a barium meal in a patient in whom we had performed an anastomosis between a loop of jejunum and the stump of common hepatic duct at the hilus of the liver (as in Figure 7) reveals regurgitation of a moderate amount of barium. Development of chills and fever led us to the decision to interrupt the proximal loop to prevent regurgitation.

the hilus of the liver. The capsule of the liver is always tough, thus, allowing application of the sutures without danger of hemorrhage or tearing. It is easier to insert these sutures before the vitallium tube is inserted into the end of the intestine; if they are placed before the intestine is put into place they can be tied readily while the tube is being held in position with the Pean forcep. The opening made in the end of the intestine to admit the forcep is then closed with one or two purse-string sutures or a few inter-

rupted sutures. We believe that some type of valve, or baffle, should be made in this arm of jejunum to prevent reflux of intestinal contents upward into the intrahepatic ducts. It is perhaps adequate to insert several interrupted sutures so as to fold the wall of the intestine into the lumen. At least two or three of these folds or valves should be made. The wound is then closed leaving a rubber drain in the upper portion. This drain, should be

FIG. 10B.—After we interrupted the proximal loop and made valves in the distal loop to further minimize regurgitation, no barium reached the intrahepatic ducts following a barium meal. The chills and fever have now disappeared. In the meantime the vitallium tube has been passed; however, it would appear that passage of the tube would have little to do with elimination of regurgitation since the patient in Figure 7 without a vitallium tube has a massive regurgitation.

left in place for four to six days since a biliary or intestinal fistula may develop any time during this interval.

(2) *Anastomosis of the Hilus Stump to a Loop of Jejunum.*—Anastomosis of a loop of jejunum to the stump of the hepatic duct at the hilus requires less operating time than an anastomosis of the end of the jejunum after the Roux-"Y" principle, as discussed above. However, three of the four cases surviving this type of operation developed chills and fever after a variable length of time following operation; one died, whereas the fifth case has only a fair result now. Therefore, we do not recommend this type of anastomosis unless an effective valve, or baffle of some type, is placed in both arms of the loop, particularly on the proximal side. The same princi-

ples described under (1) apply to this type of anastomosis. A loop of jejunum is placed in position around the colon upward toward the hilus of the liver to determine whether or not the mesentery is long enough to reach around the colon. If not, an opening must be made in the mesocolon and the loop of jejunum brought up through the opening. The funnel end of the vitallium tube is anchored in the end of the duct at the hilus with a purse-string suture, and the other end inserted in a small opening at the end of the loop and likewise anchored with a purse-string suture. Interrupted sutures are placed at three or four points between the end of the loop and the capsule of the liver around the duct before placement of the tube in the jejunum, and are tied after the tube is anchored with the purse-string suture. An entero-anastomosis must be made between the two loops of intestine, but at least 12 inches away from the anastomosis between the duct and the end of the jejunal loop. The wound is closed leaving a drain in the upper end of the wound as in the operation just described.

ANALYSIS OF CASES AND RESULTS

As stated previously, we have inserted 14 vitallium tubes in the treatment of stenosis or absence of the common duct. On ten occasions the tube was used when no common duct whatsoever could be found. In five of these cases an anastomosis utilizing the Roux-"Y" principle, as illustrated in Figures 5 and 6, was the procedure utilized. The results in four of these five patients were good to excellent. The results in the fifth case were completely clouded because of the coincidental development of a splenomegaly of the Banti's type. We do know, however, that the opening had allowed free access of bile to the intestinal tract in this patient because the stools have not been clay-colored. In the other five cases, anastomosis of a loop of jejunum to the stump of the duct at the hilus was performed (see Table II). Of these five patients, one died of hepatic insufficiency and postoperative infection. Of the remaining four, two had a fair result for two years but then began having chills and fever. The other two had fairly good results for a few months but within a year developed chills and fever. We have interrupted the proximal loop of jejunum in three of the four patients. This procedure resulted abruptly in cessation of the chills and fever in two of the cases; insufficient time has elapsed following operation in the third case to allow formulation of any conclusion regarding outcome. The fourth patient is now having chills and fever after two years of excellent result, and an operation has been recommended to sever the proximal loop.

Comparison of the results in these two groups of patients reveals that the operation utilizing the Roux arm (as in Fig. 6) is far superior to the operation anastomosing a loop of jejunum to the duct at the hilus (as in Fig. 7).

Of the four remaining cases, one had a carcinoma of the common hepatic duct; after excision of the carcinoma an anastomosis was made between the stump of common hepatic duct and the common duct over a vitallium tube,

TABLE II

CONDENSED SUMMARY OF CASES OF STRICTURE OR ABSENCE OF COMMON DUCT TREATED BY IMPLANTATION OF A VITALLIUM TUBE

Case No.	Type of Obstruction	Type of Operation	Sex Age	G. B. Removed	Repair Operation	Convalescence	Remarks	Results
1.		Duct at hilus of liver anas.	F 23	Aug. 1941	May 1943	Uneventful	Feeling fine since oper. except for epigastric pain. Had 3 chills since oper. No jaundice or acholic stools	Good to excellent
2.		to single arm (Roux - "Y") of jejunum	F 38	Dec. 1941	April 1943	Uneventful	No complaints since last oper. except one chill on 7-4-44. No jaundice	Excellent
3.		(See Fig. 6)	F 30	Feb. 1943	July 1944	Satisfactory	Since oper. has gained 40 lbs. Had a few chills early but none during past 4 wks. Still has draining sinus in wound (osteochondritis?)	Excellent (except for draining sinus)
4.			F 36	Jan. 1943	May 1943 (valves)	Uneventful	Very few complaints. Is feeling better than previous to all opers., including the first	Excellent
5.	No common or com. hep. duct found		F 39 D.S.	March 1943	April 20 and 26, 1944	Stormy. Atelectasis and stubborn distention	Large spleen found at reparative oper. has increased in size (Banti's?). Stools normal color. Still jaundiced but no evidence of duct obstruction. Ascites	Clouded by development of Banti's syndrome
6.		Duct at hilus of liver anas. to loop of je-	F 34	Dec. 1941	June 1942	Uneventful	Symptom-free for 2 yrs.; then chills and fever, without jaundice. Needs proximal loop severed	Excellent for 2 yrs. Now only fair
7.		junum (See Fig. 7)	F 38	May 1935	5 opers. 1936 to 1943	Uneventful	Four anastomoses done over a rubber tube failed. Insertion vitallium tube into loop of jejunum afforded relief for 2 yrs., then developed multiple liver abscesses and died	Excellent for 2 yrs. then chills, liver abscesses and death
8.			F 54	April 1940	May '42 Jan. '40 (valve)	Biliary and int. fistula following 2nd oper., but healed in 2 wks.	Symptoms recurred after 1st oper., because of reflux into ducts? Interruption of proximal loop to prevent reflux abolished symptoms	Excellent since 2nd oper.
9.			F 56	May 1938	Feb. '42 Oct. '43	Uneventful	Anastomosis with continuous loop of jejunum failed, but 2nd oper. interrupting ascending loop abolished symptoms	Excellent since 2nd oper. (but developed arthritis and cardiac symptoms)
10.			F 67	Aug. 1942	Jan. 1943	Postoper. wound infection. Hepatic insufficiency	Anorexia, weakness, malaise increased, with reversal of blood protein. Primary cause of death hepatic insufficiency	Died
11.	Defect or Ca. in com. hep.	Hilus duct anastomosed	F 32	Jan. 1943	Oct. 1943	Uneventful	Has had an occasional mild chill. Also some epigastric pain	Good to excellent
12.	duct. Distal com. duct found	to com. duct	F 64	None Ca. C.D.	April 1944	Developed pyloric obstruction requiring a secondary operation	Had a resection of a Ca. of C.D. Repaired duct over a vitallium tube. Few mild chills. One attack of jaundice	Good to excellent
13.			F 34	Sept. 1943	Oct. 1943	Stormy. Wound infection and peritonitis	Numerous large intra-abdominal abscesses were drained. At autopsy, many small liver abscesses found	Died
14.	Prox. and distal duct found	Com. hep. duct to com. duct	F 38	May 1942	June 1942	Uneventful	Had one attack of jaundice and acholic stools of one week's duration early after oper. Otherwise no complaints	Excellent after operation

thus, preserving the sphincter of Oddi; results have been excellent in this patient up to date (one year). In two of the four cases no common hepatic duct could be found; an operation of the type just described was performed. One patient died, but the results in the other case were good to excellent. The remaining case had a local stricture of the duct. A sizable stump of common hepatic duct and common duct were found. A vitallium tube was placed between the two stumps of the duct, although the use of the tube may not have been necessary. The results in this patient were excellent.

CASE REPORTS

Case 1.—Patient (No. 89020) was a female, age 23, who entered the Illinois Research Hospital on May 15, 1943, complaining of constant icterus following a cholecystectomy performed elsewhere, in August, 1941. There was no jaundice previous to operation. On the 14th postoperative day she had a celiotomy in an hospital elsewhere, at which time a T-tube was placed in the common duct between two cut ends. Eight months later the tube was removed; this was followed in a short time by itching and jaundice. In the meantime she became pregnant and six months later was delivered of a five-pound baby. When she entered Illinois Research Hospital she had evidence of complete biliary obstruction. Stools were completely acholic. Weakness and malaise were pronounced. The red blood count was 4.1 and the hemoglobin 80 per cent.

Operation for correction of the biliary obstruction was performed at Illinois Research Hospital, May 17, 1943. A moderate number of adhesions were encountered. These were dissected loose, uncovering the area where the common duct should be. We found one small cord which might have been a remnant of the common duct, but after considerable dissection, finally were convinced that a lumen did not exist in that area. One or two structures which we thought were portal vein were aspirated and blood obtained. The duodenum and upper margin of the pancreas were fairly easily mobilized, thereby making us more certain that no common duct remained. We, therefore, decided that we would have to find the stump at the hilus of the liver and establish an anastomosis of some type between that and a loop of small bowel. The patient was rather small and had a narrow outlet to the thoracic cage. This did not leave much room for bringing up a double loop to insert against the hilus of the liver since duodenum and liver largely filled up the right upper quadrant. We, therefore, decided that one arm of the jejunum, after the Roux principle, would be preferable to a double-armed loop. We accordingly cut across the lower jejunum and brought up the distal segment through an opening in the mesocolon and attached it to the stump of the duct at the hilus of the liver, anchoring a vitallium tube in place connecting the two structures. The intestine was anchored with three or four interrupted sutures to the hilus of the liver. The proximal end of the cut jejunum was then anastomosed to the distal loop about eight inches distal to its anastomosis with the stump of the common hepatic duct. This allowed the food to progress through the small intestine without coming in contact with the hilus anastomosis. Wound closed in layers, using interrupted cotton for the fascia, and placing a drain down to the anastomosis. The postoperative course was uneventful. Eight days after operation the icterus index was 12.

When last seen (19 months after repair) she declared she was feeling fine except for troublesome epigastric pain. During the 19-month interval she had three chills with fever, but no jaundice. Stools had been cholic at all times.

Case 2.—Patient (No. 88494) was a white female, age 38, who entered Illinois Research Hospital April 12, 1943, with the complaint of itching and jaundice. She had a cholecystectomy performed December 26, 1941, at another hospital. A biliary fistula formed and did not close until seven months later. One month after closure of the fistula she became jaundiced. After this she had recurring attacks of jaundice.

She had another operation at the same hospital February 26, 1943, following which she was free from jaundice for one month, after which time it recurred and remained constant until admittance to Illinois Research Hospital.

Operation for correction of the biliary obstruction was performed at Illinois Research Hospital, April 16, 1943. The entire gallbladder bed was found to be adherent to the upper edge of the first portion of the duodenum. This was freed with considerable difficulty. There were about 2,000 cc. of thick, bile-stained fluid in the peritoneal cavity. The region of the common duct was dissected out; the portal vein and hepatic artery were found, but there was no evidence of the common duct. Much fibrous tissue was found and explored but still no evidence of the common duct was made out. Finally, after exposing the hilus of the liver for a distance of about one-half to three-quarters of an inch, an opening was made in the stump of the duct, giving rise to a free flow of totally colorless "syrupy" bile. The funnel end of a vitallium tube was anchored in the duct with a purse-string suture of cotton; the other end of the tube was anchored in the end of the severed jejunum, as in Figure F, utilizing the Roux principle.

Convalescence was satisfactory. Two weeks after operation the icterus index was 30; it declined to normal several days later. When last seen, 20 months after operative repair, she said she was feeling fine and had gained 40 pounds. She had one slight chill July 4, 1944, but at no time had been jaundiced.

A roentgenogram, December 14, 1943, revealed the tube in place. After barium by mouth a few flakes of barium and a moderate amount of air were observed in the intrahepatic ducts.

Case 3.—Patient (No. 89009) was a white female, age 30, who entered the Illinois Research Hospital, May 14, 1943, complaining of acholic stools and a complete biliary fistula following a cholecystectomy performed elsewhere, February 5, 1943. Since operation patient also complained of pain in the right upper quadrant, loss of 25 pounds in weight and extreme weakness. There had been no jaundice previous to this operation. Laboratory examination at entrance revealed a 3-plus cephalic flocculation test, an icteric index of 117, and a red blood count of 3.7. On June 28, 1943, a celiotomy was performed but abandoned before any reparative procedure could be undertaken, because of severe hemorrhage from a tear in the liver. She recovered satisfactorily except for an increased loss of weight. She was discharged for correction of this malnutrition but returned November 11, 1943, with an acute liver abscess, which was drained as an emergency operation. During her convalescence she also developed an empyema, which required a thoracotomy. She recovered slowly and was discharged to allow improvement in her condition before the reparative operation.

Operation was performed for repair of the defect on July 10, 1944. Incision was made through the old scar dissecting out the biliary fistula which was traced down to the opening of the common hepatic duct at the hilus of the liver. No remnant of the common duct could be found except this opening at the hilus of the liver. The funnel end of a vitallium tube was anchored with a purse-string in the duct opening at the hilus and the other end implanted into the cut end of the jejunum, as shown in Figure 4. A piece of rubber tubing three inches in length was attached to the end of the vitallium tube to prevent the tube from slipping out of the end of the intestine. This arm of jejunum was brought up to the common duct under the colon. Two folds in the jejunum were made in an attempt to duplicate valves. Wound closed with through-and-through sutures. Three Penrose drains left in place. The postoperative course was uneventful and afebrile. The icterus index was 11 at time of discharge on the 14th postoperative day. Culture of bile made at operation revealed *alpha Streptococcus*, *B. coli*, *B. hemolyticus Streptococcus* and diphtheroids.

Patient has gained 40 pounds since operation. Up until December, 1944, she had eight chills with fever but no jaundice. When last seen, in March, 1945, she had had no chills for four months and was feeling fine. However, she has a draining sinus

which is probably caused by a chondritis or osteomyelitis secondary to the liver abscess. A roentgenogram, taken in March, 1945, revealed no tube, indicating that it had passed.

Case 4.—The patient was a white female, age 36, who gave a history of having had a cholecystectomy performed in an hospital elsewhere, in January, 1943. Within a few days she became jaundiced and had clay-colored stools. Jaundice persisted constantly for several weeks, after which time she had another celiotomy. At this time, a dense mass of fibrous tissue was found in the region of the common duct, but no trace of the duct itself could be found. Jaundice and clay-colored stools persisted.

Operation for repair of the duct obstruction was performed in May, 1943. A moderate number of adhesions were encountered between the surface of the liver and contiguous organs. Separation of these adhesions down to the posterior peritoneum revealed no trace of the common duct. Aspiration of the only tubular structure in that neighborhood yielded blood, indicating that it was the portal vein. The hilus of the liver was then explored and a slightly bulging point aspirated; thin, slightly bile-stained fluid was obtained. This stump of common duct was opened and the funnel end of a vitallium tube anchored in place with a purse-string suture of silk. The other end of the vitallium tube was anchored in the cut end of the jejunum, as in Figure 6. An attempt was made to create valves in the arm of jejunum by making two folds of the intestinal wall with interrupted silk sutures. The arm of jejunum was brought up to the hilus through an opening in the mesocolon. Wound closed leaving a drain in the upper end. Convalescence was uneventful. Stools became cholic and remained so up to date. Patient had a few mild chills soon after the plastic repair, but declares that she feels better now than she did before any of her operations, including the first one.

Case 5.—Patient (No. 99027) was a white female, age 39, who entered the Illinois Research Hospital complaining of jaundice and clay-colored stools. She gave a history of having had a cholecystectomy, in March, 1943, at a hospital elsewhere. The gall-bladder was filled with many stones. She had a stormy postoperative course due to wound infection and rupture of the incision. About four months after the cholecystectomy patient noticed jaundice which became progressively worse up until time of entrance in Research and Educational Hospital. Itching and clay-colored stools likewise were present. Examination revealed deep jaundice. A large mass occupied the entire left upper quadrant extending to the iliac crest; it was presumably an enlarged spleen. Liver edge was palpable 4 cm. beyond costal margin. Stools were acholic. The icterus index was 78, and the cephalin flocculation 3 plus. The N. P. N. was 24 mg. per cent and the A/G ratio 3.4/2.6 mg. per cent. The red blood count was 2.5, requiring several transfusions before patient could be considered operable.

Operation was performed for correction of the duct obstruction, April 20, 1944. Incision was made through the old scar. The abdominal wall was unusually vascular. After entering the peritoneal cavity numerous adhesions were encountered which likewise were very vascular. This made it appear definite that there was a portal hypertension, supported by the fact that the spleen was enormously enlarged. The liver itself was markedly enlarged, dark red in color and congested. Perhaps 40 minutes were consumed dissecting colon, duodenum, *etc.*, away from the hilus of the liver before we could obtain exposure in that area, which was badly "frozen," and all structures were covered with so much fibrous tissue that landmarks were very poorly visible, if at all. No trace of a common duct could be found. There were two large structures going toward the hilus of the liver. They were poorly defined because of fibrous tissue, but aspiration yielded blood, indicating that they were veins, although the dual structure at this point was difficult to explain except by anomaly of premature branching of the portal vein. Both of the venous structures were somewhat indurated, suggesting that there was, or had been, a thrombosis, although the aspiration of blood indicated that they were recanalizing. By continued aspiration we encountered an area in the hilus of the liver where bile-stained fluid was obtained. This was slightly to the right

of the two veins and artery, but aspiration directly to the right of the area likewise yielded blood. This area leading to the hepatic duct was apparently entirely surrounded by large veins, but by utilizing unusual care we got into this cavity without damaging any of the branches of the portal vein. Further dissection downward toward the duodenum revealed a thin, small structure which was obviously the remnants of the common duct. It was filled with plastic exudate, and appeared now to be entirely blocked. The mucosal surface appeared to be destroyed, and it seemed likely that it would ultimately, quite definitely be completely stenosed. It appeared then that we would have to make an anastomosis of the hilus of the liver to a loop of intestine. In our endeavor to dilate the opening in the hepatic duct, we damaged a sizable branch of the portal vein within the liver which made it impossible to continue. We, therefore, decided to pack this area, and if bile began to flow postoperatively we could go back in several days later, and perform the anastomosis. Accordingly, a small gauze pack was placed firmly against the hilus of the liver, and the wound closed around a drain left in the upper portion.

The pack was removed on the third day. This was followed by a gush of bile, which drained constantly making a second operation seem justifiable.

Second Operation was performed, April 26, 1944, hoping to be able to complete the anastomosis of the hilus to a loop of intestine. Since insufficient time had elapsed to allow formation of adhesions, we obtained exposure of the hilus of the liver without much difficulty. The opening of the common hepatic duct at the hilus was dilated and the funnel end of a vitallium tube anchored in with a purse-string suture of silk. We implanted the other end in the severed end of the jejunum, after the Roux principle as illustrated. The arm of the jejunum was brought up through an opening made in the mesocolon. The liver was so swollen and congested that it seriously obscured our field at the hilus, and the vitallium tube was actually too short to bridge across the area between the mucosal surface of the hepatic duct and the mucosal surface of the intestine. We accordingly spliced a rubber tube two inches long onto the end of the vitallium tube. This allowed us to anchor the end of the intestine against the hilus of the liver without any possible danger of the vitallium tube slipping out of the intestine. We attempted to produce a valve in the arm of jejunum by infolding the wall acutely into the lumen with several interrupted cotton sutures. The wound was closed in layers around a Penrose drain in the upper portion.

The patient's convalescence was stormy because of atelectasis and a stubborn distention which responded poorly to intestinal decompression.

The stools became cholic, but the splenomegaly and jaundice persisted. The spleen, in fact, was enlarged much more now than at the time of operation, and an ascites had developed. The entire picture of splenomegaly, portal hypertension and ascites strongly suggested the coincidental development of Banti's syndrome. Since this is the only case of its type in our series we assume the clinical picture is coincidental unless it is related to the portal thrombosis which we are certain was present even at the time of the first of the two reparative operations.

Case 6.—Patient (No. 83352) was a white female, age 34, who entered Research and Educational Hospital June 10, 1942, with a history of having had a cholecystectomy December 30, 1941, at a hospital elsewhere. Considerable bleeding around the junction of the cystic and common duct required the insertion of an artery forcep, which was left on for a day or two. A few days after operation an external biliary fistula developed. Considerable purulent discharge likewise drained from the fistula for several days. Shortly thereafter jaundice accompanied by frequent chills with fever developed.

Operation for correction of the duct obstruction was performed at Illinois Research Hospital, June 13, 1942. Numerous adhesions were present. No trace of the common duct was found. Accordingly, a loop of jejunum was brought up through an opening in the mesocolon and attached to the hilus of the liver over a vitallium tube, the funnel end of which was anchored in the stump of the common hepatic duct with a purse-

string suture of silk. Illustrated. Wound closed around a Penrose drain in the upper portion.

Convalescence was uneventful. Following discharge from the hospital she was symptom-free for two years, after which time she began to have frequent chills accompanied with fever. On one occasion she developed slight jaundice which disappeared in a few days. A roentgenogram showed the tube in place, but administration of barium by mouth revealed a massive reflux of barium into the intrahepatic bile ducts.

COMMENT: Since the tube is still in place (according to the roentgenogram), thus, preventing any stenosis from developing at the anastomotic line, the only explanation for the chills appears to be a cholangitis, which could readily be caused by the reflux of intestinal contents into the intrahepatic bile ducts. Another operation to interrupt the proximal limb of the jejunal loop and perhaps construction of folds in the distal descending loop of jejunum for valves, appears justified, and has been advised.

Case 7.—Patient (No. 81941) was a white female, age 38, who entered Illinois Research Hospital, March 15, 1938, complaining of jaundice and severe itching. She had a cholecystectomy performed in May, 1935, at an hospital elsewhere. She remained well for five months, after which time she developed epigastric pain, nausea, vomiting and, later, jaundice. In February, 1936, celiotomy was performed at another hospital, at which time the duodenum was anastomosed to the stump of the common hepatic duct at the hilus of the liver, over a rubber tube. Several months later she passed the rubber tube and again became jaundiced. At Illinois Research Hospital she had another anastomosis performed, April 6, 1938, between the duodenum and stump of the common hepatic duct over a rubber tube. Eight months later, jaundice and other symptoms recurred. On January 17, 1941, the same operation as described above was again performed. However, several months later symptoms recurred. On March 18, 1942, we sutured the end of the common hepatic duct at the hilus to a loop of jejunum over a rubber tube. The loop of jejunum was brought up to the hilus through an opening in the mesocolon; an anastomosis between the two arms of the jejunum was made two or three inches inferior to the mesocolon, hoping to shunt the food away from the suture line at the hilus. Patient was well until five months later, when she passed the rubber tube, after which she became jaundiced and had numerous chills with fever. On March 31, 1943, she was operated upon again and a vitallium tube placed between the stump of the common hepatic duct and loop of jejunum. Roentgenologic studies following barium by mouth 1.5 years after the reparative operation revealed the intrahepatic ducts filled with barium. A few weeks later a roentgenogram revealed the absence of the vitallium tube; which had obviously been passed. She remained entirely well for two years, then suddenly developed severe chills with fever. After 12 days observation, and unsuccessful treatment with penicillin and sulfadiazine, we operated upon her, interrupting the proximal loop (which was allowing reflux of food) hoping that the infection still was limited to a cholangitis and had not progressed to multiple abscess formation. However, the infection had apparently reached the hopeless stage since she died a few days later. Autopsy revealed multiple abscesses of the liver.

Case 8.—Patient (No. 82624) was a white female, age 54, who entered Research and Educational Hospital, April 28, 1942, complaining of jaundice and clay-colored stools. She gave a history of having had a cholecystectomy in April, 1940, at an hospital elsewhere. Before cholecystectomy patient had epigastric pain, nausea, vomiting, belching and bloating, but no icterus. She developed jaundice on the second postoperative day, which became progressively worse. She was reëxplored two weeks after cholecystectomy; an external biliary fistula was established which drained for three months. Following this, the wound healed. However, recurrent jaundice occurred with chills, fever, pain and swelling in right upper quadrant. These symptoms would disappear

when external biliary fistula recurred. Attacks occurred one to three times monthly and lasted for two to five days.

Operation was performed at Illinois Research Hospital for correction of the duct obstruction, May 4, 1942. There were numerous adhesions over the entire upper quadrant following the previous operations. The gallbladder was gone. We followed the liver edge from the outside, working medially. We came to an attachment of the colon with the hilus of the liver. When this was cut we noted a small opening in the colon suggesting there had been a fistula between the colon and the common duct. Drainage of a small amount of bile from the liver side of this attachment offered further support that there was a fistula between the common hepatic duct and colon. It was, of course, impossible to determine what the previous operations had to do with this fistula. We tried to find the distal end of the common hepatic duct or any portion of the common duct. We were unsuccessful. We then opened the duodenum low in its second portion to see if we could find the sphincter of Oddi, and probe the ducts from the duodenal side. After several minutes exploration we were unable to find the ampulla of Vater. We then closed the opening in the duodenum and decided to bring up a loop of jejunum and perform an anastomosis between it and the hepatic duct at the hilus over a vitallium tube. We inserted the funnel end of a small-sized tube in the hepatic duct and closed the opening around it with a purse-string suture of silk. We then made an opening in the loop of jejunum and inserted the other end of the tube. We then established an anastomosis between the two loops of jejunum about six inches away from the anastomotic line. This shunted the food away from the anastomosis between the duct and the jejunum. We had brought the jejunum up through a hole in the mesocolon. The wound was then closed, leaving a drain in the upper portion.

Convalescence was uneventful and the patient was free from symptoms for a few months. However, after that period she developed recurrent attacks of chills, fever and icterus. Roentgenologic studies, January 25, 1944, following barium by mouth showed a barium fill-up of the intrahepatic ducts. We, accordingly, decided to operate upon the patient again, and interrupt the proximal loop of jejunum hoping to exclude intestinal content from the biliary ducts.

Second Operation.—January 28, 1944: The anastomotic opening between the two loops of jejunum just under the mesocolon was found to be ample. We then resected about three inches of the proximal loop, thus, interrupting the bowel so that food could not go around the normal channel past the hilus of the liver. To further prevent influx of food upward toward the vitallium tube and hilus of the liver, we made a valve in the distal or descending loop. The valve in the intestine was made by making a horseshoe-shaped incision with the base toward the hilus and inverting this flap so that there would be a valve-like mechanism preventing food from going upward, but allowing bile to drain downward. When the opening was closed we were a bit concerned as to whether or not our valve flap was too large, but thought we would take the chance since we knew a certain amount of atrophy and contracture of the flap would take place. Culture of bile taken at the time of operation from the intrahepatic ducts revealed *Staphylococcus albus, B. proteus, B. coli* and *gamma Streptococcus.*

Postoperatively, she developed a biliary fistula which appeared to include a small quantity of intestinal content, but no food particles. Apparently we had created a temporary obstruction to the bile with our large flap. However, after 10 or 12 days the fistula closed and the wound healed.

When last seen, in April, 1945, she was feeling fine and had had no symptoms since her last operation 15 months previously. However, a roentgenogram, February 8, 1945, revealed no vitallium tube; apparently it had passed. No barium refluxed into the intrahepatic bile ducts but a small quantity of air was visible.

Case 9.—Patient (No. 86931) was a white female, age 56, who entered the Research and Educational Hospital, January 29, 1942, complaining of jaundice, chills and acholic stools. At another hospital she had had a cholecystectomy in May, 1938. Patient was

jaundiced prior to cholecystectomy which did not ameliorate the jaundice. Icterus became more pronounced with loss in weight, *etc.*, later. In January, 1940, she had another operation at which time no common duct could be found. A catheter was sewn into the common hepatic duct at the hilus to create an external biliary fistula. Convalescence was stormy. Six months later she was operated upon again (likewise, at another hospital) and a T-tube sutured in place between the duct at the hilus and a loop of jejunum. Recovery was slow, but after a few weeks the jaundice disappeared. In November, 1940, the T-tube was removed because of discomfort. She remained well until July, 1941, after which time jaundice, chills, fever and malnutrition returned.

Examination upon admission to Illinois Research Hospital revealed jaundice and moderate malnutrition. The red blood count was 4.3.

Operation was performed for correction of the obstruction, February 4, 1942. Numerous adhesions were present, which after separation revealed a loop of jejunum attached to the hilus of the liver, where it had been placed at a previous operation done elsewhere. There was an anastomosis between the two loops of jejunum down low, so that food could pass without going around the loop over the duct anastomosis. Cutting across the anastomosis between the duct at the hilus of the liver and the jejunal loop revealed a total stricture of the common duct at this point. Bile began to flow freely. A rubber tube, two inches long and size No. 18, was inserted at this anastomotic junction and anchored with two stainless steel sutures and two of cotton against the hilus, hoping to keep it in position. The wound was closed around a drain in the upper portion. The postoperative course was uneventful. Patient remained symptom-free for only seven months and returned with chills and fever.

Second Operation.—January 15, 1943: A small incision was made at the junction of the loop of jejunum and the duct at the hilus and a vitallium tube was inserted, with the funnel end in the bile duct and the other end in the jejunal lumen. The rubber tube had been passed. A drain was left in the upper end of the wound.

She remained symptom-free for about six months, and again returned with jaundice, chills and fever. However, her stools were always cholic, indicating that there was a patent opening for entry of the bile into the intestinal tract. A roentgenogram following oral administration of barium revealed a free reflux of barium into the intrahepatic bile ducts; this tended to confirm our suspicions that the cause of the chills was a cholangitis.

Third Operation.—October 2, 1943: Interrupting the ascending or proximal loop of intestine, but not making any valves in the descending or distal loop. The jaundice, chills, fever and anorexia disappeared in a week or so and have remained absent to date (March 6, 1945) with the exception of three or four chilly sensations she had in October and December, 1944. However, in the meantime she had developed a diffuse and rather severe arthritis along with certain cardiac symptoms including precordial pain, dyspnea and ankle edema. There is no evidence that these symptoms are related to her biliary difficulties. A roentgenogram following oral barium still reveals a reflux of barium into the intrahepatic ducts, resulting from our failure to place valves in the remaining arm of jejunum; however, the interruption of the proximal loop has apparently minimized the reflux of intestinal content into the bile ducts sufficiently to prevent infection, *i.e.*, cholangitis.

Case 10.—Patient (No. 85338) was a white female, age 67, who entered Illinois Research Hospital first in November, 1942, with the history of jaundice, acholic stools and a biliary fistula since a cholecystectomy performed in August, 1942, at a hospital elsewhere. Shortly before the patient entered Illinois Research Hospital she developed a right femoral thrombosis, with pronounced edema of the entire extremity. Anorexia, weakness and malaise were so pronounced and responded so feebly to therapy that we sent her home for a short time hoping that home environment would improve her.

However, at a second admission, late in December, 1943, the patient was still found to be a poor surgical risk; anorexia and weakness were persistent. Her condition improved but little, with transfusions, forced feeding and parenteral feeding of glucose

and amino-acids. When it appeared that we had obtained as much improvement as possible we subjected her to a celiotomy (January 8, 1943).

Operation.—No common duct was found except for a stump of the common hepatic duct at the hilus of the liver to which the biliary fistula led. We, accordingly, brought up a loop of jejunum through an opening in the mesocolon and anastomosed it to the short stump of the common duct over a vitallium tube; an anastomosis was made between the two loops of jejunum inferior to the mesocolon.

Convalescence was stormy and complicated by a wound infection. The blood protein became reversed (alb. 2.4, glob. 3.3 Gm. per cent) and the patient was apathetic, indicating the probable presence of a severe hepatic insufficiency. The weakness and malaise increased in spite of transfusions, *etc.;* and she died three weeks after operation. At autopsy, the most significant finding was an extensive cellular necrosis of the liver, although a small subphrenic abscess was also present.

Case 11.—Patient (No. 96826) was a white female, age 34, who entered the Research and Educational Hospital, September 8, 1943, complaining of jaundice and pain in the right upper quadrant. She had had a cholecystectomy and choledochostomy performed in January, 1943, at a hospital elsewhere. She was well for three months, after which time she developed itching and jaundice and a recurrence of her pain in the right upper quadrant. Examination revealed jaundice (icterus index 26) and acholic stools.

Operation was performed at Illinois Research Hospital for repair of the duct obstruction, October 1, 1943. Numerous adhesions were present in the right upper quadrant; after their separation a bulging stump of proximal common hepatic duct, measuring 2 or 3 cm. long, was found. A stricture was present at the terminal end of this stump. Further dissection revealed the terminal end of the common duct, with a defect of about 1 or 1.5 cm. intervening. A probe was placed in the distal end. It passed readily down to the duodenum. However, we could not be certain that it passed into the duodenum. There was so much uncertainty that I finally opened the duodenum to clear this point. We found that the probe came up against the duodenal wall, but nowhere could it be made to enter the duodenum. There was obviously a stricture at this point as well as higher up. We incised the duodenal wall over the probe, and dilated this fibrotic area. We then took the end of a catheter (size No. 16) and threaded it up into the distal end of the common duct, allowing about three inches to remain in the lumen of the duodenum. We then closed the opening we made in the duodenum and placed a vitallium tube (with a flange in the center but no funnel tip) between the two ends of the common duct at the constriction. We could not bring the two ends together without jeopardizing the blood supply, but were able to close over the defect with adjacent tissue. Wound then closed in layers, using interrupted cotton for the fascia.

Convalescence was uneventful. However, she complained of considerable epigastric pain and an occasional mild chill. It was thought that the epigastric pain was due to a ventral hernia which had developed following the first operation and had been repaired inadequately at the second operation on account of the duration of the operative work for correction of the duct obstruction. Accordingly, several months ago the hernia was repaired. The vitallium tube was found in place and covered so completely with fibrous tissue that it was not disturbed.

A roentgenogram, February 16, 1945, showed the tube in place. After barium by mouth no barium or air was noted in the intrahepatic ducts.

Case 12.—Patient (No. 93977) was a white female, age 64, who entered Illinois Research Hospital, April 18, 1944, complaining of jaundice and acholic stools of six months' duration, with associated pain of only mild degree. She had not had any previous operations.

Operation.—April 24, 1944: Celiotomy disclosed a distended gallbladder; the head of the pancreas was normal; and the distal end of the common duct collapsed. A carcinoma was present in the common duct at the junction of the cystic duct, extending up

toward the hilus of the liver but not into it. There was enough uninvolved common hepatic duct to allow us to accomplish a local resection. A segment of normal duct about one-half inch long was left on each side of the specimen. The duodenum mobilized readily, allowing us to bring up the common duct to meet the cut end of the hepatic. Exploration of the distal end of the common duct revealed a doubtfully patent outlet at the sphincter of Oddi. We opened the duodenum over the sphincter and discovered that the probe came up against the duodenal wall but there was no sphincter opening. We, accordingly, cut down upon the probe and inserted a piece of rubber tubing two inches long, upward into the terminal end of the common duct, leaving about three-quarters of an inch protruding into the lumen of the duodenum. The opening in the duodenum was then closed. An anastomosis was performed between the two cut ends of the common duct around a vitallium tube without tension. Interrupted cotton was used for the suture material. Previous exploration had revealed no metastasis anywhere. Wound closed around a Penrose drain, using interrupted cotton for the fascia.

Convalescence uneventful for few days but patient then developed a pyloric obstruction which would not subside with decompression. We, therefore, reoperated on May 18, 1944. Very dense adhesions were found in the right upper quadrant. They were so dense that we assumed dissection of the pylorus and duodenum from this mass might result only in temporary relief. We accordingly performed a posterior gastro-enterostomy.

Recovery was uneventful. Since discharge, she has had a few mild chills and one attack of jaundice, without fever, which, however, cleared after two weeks, indicating that it was not due to metastases. Since then she has had very few symptoms, although insufficient time has elapsed to determine the prognosis from the standpoint of metastases. She did not pass the rubber tubing which we had inserted upward into the distal end of the common duct until eight weeks after insertion.

A roentgenogram, February 13, 1945, showed the vitallium tube in place. After barium by mouth no barium or air was found in the intrahepatic ducts.

Case 13.—Patient (No. 91465) was a white female, age 34, who entered Illinois Research Hospital in October, 1943, complaining of jaundice and acholic stools which had been present before and since an operation performed elsewhere six weeks previously. The operation in September, 1943, had not relieved the common duct obstruction.

Operation was performed October 28, 1943, at Illinois Research Hospital hoping to correct the obstruction. A defect was found in the common duct; no proximal segment could be found, but a distal segment was isolated. The duodenum and distal end of the common duct were mobilized and distal end of the common duct sutured to the stump of the common hepatic duct at the hilus, over a vitallium tube.

A culture of bile taken at time of operation revealed *Esch. coli, gamma Streptococcus* and *Staphylococcus albus.* Convalescence was stormy. Fever was present, and appeared to be explained, only in part, by a wound infection which developed a day or two after operation. Signs of peritonitis developed which were controlled only to the point of localization by intravenous sodium sulfadiazine. Numerous abscesses developed. On November 8, 1943, a cul-de-sac abscess was drained; on November 15, 1943, an abscess in the left upper quadrant; on December 3, 1943, an abscess in the neighborhood of the wound, and on December 13, 1943, a liver abscess was opened. In spite of this, however, her condition became worse and she died eight weeks after operation. Autopsy revealed numerous abscesses in the liver.

COMMENT: The patient had had fever for several days before operation, and the usual means of treatment including the use of sulfonamides were not effective in eliminating the infection, which presumably was a suppurative cholangitis. In retrospect, it appears it would have been better not to have proceeded with the complete operation in the presence of the infection and fever; no doubt, we should have established better drainage of the bile ducts,

with the aid of a catheter and drains, as a preliminary operation, thereby hoping to eliminate the infection before imposing the load of the entire reparative operation upon the patient. Penicillin was not then available.

Case 14.—Patient (No. 83167) was a white female, age 38, who entered Research and Educational Hospital May 29, 1942, with a history of recurrent attacks of icterus, clay-colored stools and cramping pain in the right upper quadrant for one year preceding cholecystectomy, performed May 7, 1942, at a hospital elsewhere. Patient has been jaundiced continually since second postoperative day; in addition, she has had itching, clay-colored stools and an external biliary fistula. The icterus index was 87. The red blood count was 3.1, and the hemoglobin 11 Gm. per cent.

Operation was performed at Illinois Research Hospital for a biliary fistula and obstruction of the common duct, June 4, 1942. A stricture of the common duct was found. It was not possible to bring the ends of the duct together; therefore, a vitallium tube was sutured into the duct with the funnel end inserted proximally. One Penrose drain was placed down to the vitallium tube; and the wound closed in layers.

Recovery was good, with practically no complaints except for one attack of jaundice associated with acholic stools two years postoperatively. This attack of jaundice lasted for one week, but subsided completely; and patient has had no difficulty since.

A roentgenogram, December 20, 1944, showed the tube in place. After barium by mouth no barium or air was found in the intrahepatic ducts.

COMMENT: The chief advantage of vitallium tubes in the operative repair of strictures of the common duct is to prevent a recurrence of the stricture which so commonly happened after the old conventional method of repair. Commonly, a rubber tube was used but there is a great deal of evidence supporting the fact that rubber is much more of a foreign body than is vitallium. In the repair of complete absence of common duct, an anastomosis of the stump of the common hepatic duct at the hilus of the liver to the functioning duodenum, with implantation of a vitallium tube to maintain patency of anastomosis, would appear to be the simplest type of repair. However, the authors have been of the opinion that the food stream would sooner or later dislodge the vitallium tube which would naturally be protruding into the lumen of the functioning intestine. Accordingly, we have not used this type of repair. It appears to us that one of the greatest advantages of the tube would be in its continued presence at the anastomotic line, thus, eliminating stricture formation. However, as will be discussed below, there is opportunity for reflux of food and intestinal secretions through the tube into the intrahepatic ducts which would be a source of considerable danger from the standpoint of cholangitis.

The pathologic lesion producing the chills and fever in stricture of the common duct is cholangitis. In a stricture of the common duct, with the sphincter of Oddi normally intervening between it and the intestine, obstruction would appear to be the primary factor in pathogenesis of the cholangitis. However, when correction by an operative procedure attaching the intestine directly to the duct has been performed, thus, eliminating the protective action of the sphincter, an added etiologic factor (*i.e.*, reflux of food and intestinal content) is introduced. We are convinced that on certain occasions the

reflux of food and intestinal content is more important than obstruction. For example, Figure 9 illustrates reflux of barium through the anastomosis performed between the hilus stump and the duodenum after the conventional method. The patient has had numerous attacks of chills and fever, but has never had more than latent jaundice, implying that any obstruction present is at least too insignificant to produce jaundice. It seems obvious that if the opening was large enough to allow reflux of barium, it should be large enough to allow bile to flow through it, but, likewise, it would be large enough to allow reflux of food and intestinal secretions up into the liver with consequent development of cholangitis. It might be claimed that the chills and fever in the case just mentioned were due to partial obstruction. However, if partial obstruction was the primary factor in the production of cholangitis of this type it would appear that we should have chills and fever more consistently in patients with obstruction (partial or complete) of the common duct by stone. Although chills and fever do occur in obstruction of the common duct by stone the condition is, nevertheless, relatively uncommon. In our opinion, the sphincter of Oddi represents a protecting mechanism in a common duct obstructed by stone. Accordingly, it would appear that we should attempt to create the effect of the sphincter of Oddi in our repair of strictures of the common duct, although we grant that it is impossible to duplicate it exactly. The logic of this statement is supported by the fact that in our small series we had good to excellent results in all patients in whom we were able to preserve the function of the sphincter of Oddi.

Another factor which convinces us that reflux of intestinal content is important in the development of cholangitis is the poor result in four patients surviving the operation of anastomosis of the stump of the common hepatic duct at the hilus to a loop of jejunum, as in Figure 7. It is true that two of these patients had good results for a year or two, but, ultimately, chills and fever without jaundice recurred. Examination during a gastrointestinal series revealed that there was a reflux of barium through the vitallium tube into the intrahepatic duct in all four cases. Although we had performed a short-circuiting anastomosis between the two loops of jejunum at some distance from the anastomosis, a great portion of the intestinal stream continued to follow the normal channel rather than go through the anastomotic stoma. Three of these patients have been reoperated upon, and the proximal limb of jejunum interrupted, thereby preventing gross reflux. Two of these have had no chills since this operation, the third was just operated upon and insufficient time has elapsed to determine results.

In our experience with the two types of jejunal attachment we are, therefore, convinced that the attachment of a single arm of jejunum, after the Roux principle, to the hilus stump is a much better operation than anastomosis of the duct stump to a loop of jejunum. This arm of jejunum must be at least 24 inches long, since we have already learned that regurgitation can take place in short loops, particularly if valves or baffles have not been created.

We have had so many instances of cholangitis following various types of plastic operation upon the common duct that we have come to fear it more than any other complication. It has long been known that cholangitis is a serious complication, insofar as multiple abscesses of the liver are so apt to develop. We have sufficient evidence that cholangitis of the type presented in this kind of obstruction is of bacterial type, because cultures made of bile on every occasion when we have taken them at the operating table in these stricture cases have been positive. Invariably, the flora is a mixed one, consisting primarily of *B. coli,* streptococcus and staphylococcus. one of these patients developed an uncontrollable cholangitis followed by multiple abscesses of the liver, and died a few weeks after operation. In our limited experience, cholangitis has been uncommon in plastic repair of the duct when it has been possible to preserve the function of the sphincter of Oddi; it has been relatively uncommon when sufficient common hepatic duct is available to transplant into a loop of intestine (usually duodenum) as we have done in stricture due to pancreatitis, and resection of the head of the pancreas for carcinoma, not herein reported. However, it has been encountered frequently in certain types of operation when the common as well as common hepatic duct were missing.

We are not yet convinced that the vitallium tube needs to stay in permanently, although it appears desirable. The flange on the shaft of the tube was placed there primarily to prevent the tube from slipping out of position. The flange and the funnel exert a definite influence in maintaining position of the tube, but of the 14 tubes implanted by us, three became dislodged and were passed. If the tube remains in for several weeks or months and is then passed, we believe that the anastomotic opening must be sufficiently large to prevent significant stricture formation. Two of the patients in whom the tube was passed are completely free from symptoms of cholangitis, but only after we interrupted the jejunal loop so that reflux of barium, and presumably food, into the intrahepatic ducts could not take place. The third patient who lost the tube was one who was having reflux of barium, and presumably food, into the liver following the less desirable operation of anastomosing the hilus stump to a loop of jejunum. She developed serious chills, and later died, but we do not believe the passage of the tube had anything to do with this since reflux of food into the liver was taking place and would have done so even if the tube had remained in place.

SUMMARY AND CONCLUSIONS

We have reviewed our experience with 23 patients with stricture or absence of the common duct, including two cases of carcinoma of the common hepatic duct. In the repair of these defects we utilized implantation of a vitallium tube in 14 cases except for indefinite fibrous bands. In ten of the 14 we were unable to find any trace of the common duct. In these ten cases we anastomosed the stump of the common hepatic duct to a single arm of the jejunum (utilizing the Roux principle) in five instances, and anastomosed the

duct stump to a loop of the jejunum in five instances. In general, the results were good to excellent in patients having anastomosis between the duct stump and arm of jejunum. On the contrary, the results were poor in the cases in which we anastomosed the duct stump to a loop of jejunum which allowed regurgitation of barium, and presumably food, in spite of an anastomosis, which we had performed between the two loops at some distance from the anastomosis with the duct. Interruption of the proximal arm of jejunum, which was presumably allowing the reflux of food through the tube or duct anastomosis into the liver, in general, has obliterated the recurrent chills and fever. Obstruction unquestionably is an important factor in the development of cholangitis with chills and fever in the failure of plastic operation upon the common duct. However, *prevention of obstruction by the use of a tube,* in our experience, *did not prevent the occurrence of chills and fever unless we prevented reflux of food by operative procedure.* We have come to the definite conclusion, therefore, that the best operation in the type of patients in which no common duct can be found, is anastomosis of the stump of the hepatic duct to a single arm of the jejunum which is at least 24 inches long and the walls of which have been folded to produce valves or baffles.

In four patients, the terminal end of the common duct was found, thereby allowing us to preserve the sphincter of Oddi. Although one of these patients died following operation, the other three had good to excellent results. We attribute this largely to the fact that the sphincter of Oddi, with its normal function, prevents reflux into the duct.

In the 14 cases we had two operative deaths, constituting a mortality rate of 14.3 per cent. This can be lowered appreciably if we do not operate upon patients who are known to be very poor risks. We knew that one of the two patients who died was an exceedingly poor risk, but since restoration of bile to the intestinal tract seemed to be the only hope of obtaining recovery we subjected her to operation.

In our opinion, the vitallium tube has a definite place in reconstruction in strictures and absence of the common duct, but primarily in the patients in whom no common duct whatsoever can be found, and in the group in which the common hepatic duct cannot be found.

We believe that in local strictures the first operation should consist of anastomosis of the two ends over the arms of a T-tube inserted a short distance below the line of suture; if a stricture forms at the site of anastomosis then a vitallium (or tantalum) tube should be implanted later.

Cholangitis is sufficiently infrequent following implantation of a stump of common duct into the intestine to justify this procedure without use of a tube; if cholangitis does develop, consideration should be given to a second operation and use of a tube. However, whenever a tube is used it should be implanted into a nonfunctioning single arm of jejunum (as in the Roux procedure) to prevent reflux of food and prevent the food stream from dislodging the tube.

We have had opportunity to observe a few tubes after they had been in

place for a year or two, but in none of those seen was there evidence of corrosion or precipitation of bile salts on the wall of the tube.

We have not used tantalum, but this metal should have advantages over the alloy vitallium, insofar as it is pliable and can be bent or cut at any desired length; however, a flange or funnel end, or both, would be necessary to maintain the tube in position.

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ADVANCED CARCINOMA OF THE EXTRAHEPATIC BILE DUCTS: CHOLEANGIOCHOLECYSTOCHOLEDOCHECTOMY*†

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EXTENSIVE INVOLVEMENT of the extrahepatic biliary tract by carcinoma is one of the most hopeless situations confronting the surgeon. The anatomic relationships of these ducts renders impossible a wide excision of the growths, and operations in this region are difficult because of the portal vein and hepatic arteries which are in the field, and are often partially or extensively surrounded by neoplasm. The subject of carcinoma of the extrahepatic bile ducts was thoroughly reviewed up to 1940, by Stewart, Lieber, and Morgan. A critical analysis of the reports in the literature, most authors reporting one or two patients, showed that surgical therapy was instituted in 50 cases. The ultimate mortality rate was 98 per cent, the fate of one patient remaining unrecorded. The immediate postoperative mortality was 68 per cent. Table I is a summary of statistics from this study:

TABLE I

SUMMARY OF 50 CASES OF EXTRAHEPATIC BILE DUCT CARCINOMA

	Carcinoma Hepatic Duct	Carcinoma at Confluence of Extrahepatic Bile Ducts	Carcinoma Common Bile Duct
No. of cases treated surgically	21	21	8
Avg. survival	6.5 mos.	7.2 mos.	2.2 mos.
Avg. survival with "medical" treatment	8.2 mos. (12 patients)	4.6 mos. (26 patients)	4.4 mos. (13 patients)

In eight case reports[2–6] of advanced carcinoma of the extrahepatic biliary tract in the more recent surgical literature simple exploration with attempted drainage was carried out in five instances, with average postoperative survival of nine days. In the remaining three instances one was discharged 48 days after drainage, one survived resection for 11 months, having received good palliation for five months, and one was well seven months after operation in which T-tube reconstruction of the common duct had been performed.

From the above results it appears that surgical intervention which in most instances was simple exploratory celiotomy, with or without attempts at drainage of the biliary tract, afforded little palliation in most instances.

In view of this pessimistic picture, a series of seven patients presenting advanced carcinoma involving most of the extrahepatic bile passages and extending into the gallbladder, and in whom there was no apparent diffuse peritoneal spread or hepatic metastases, were subjected to radical resection of these ducts and the gallbladder, sometimes with excision of liver tissue about

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the gallbladder and in one instance resection of a portion of the head of the pancreas. The purpose envisaged was to ascertain if palliation might be afforded where most, or all, of the macroscopic neoplasm was removed. Drainage by simple insertion of a catheter high in the extrahepatic biliary tract was the only other alternative, and this did not appear to be a satisfactory procedure to carry out, because incision and probing into dense tumor tissue would have been necessary and would have been difficult to perform since the ducts high in the porta were involved in a dense contracted mass.

A summary of the patients and results are as follows:

SUMMARY OF RADICAL OPERATION UPON SEVEN PATIENTS WITH EXTRAHEPATIC BILE DUCT CARCINOMA

Case 1.—S. P. (No. 264586), male, age 67.

Carcinoma involving extrahepatic bile ducts, gallbladder, its liver bed, and the upper right lateral portion of head of pancreas. Survived operation for one year. Icterus completely cleared; return to full-time normal activity for several months; gain in weight. Second operation performed when icterus recurred 11 months after first operation. Vitallium tube inserted, *into dense mass of recurrent carcinoma in porta*, but condition did not improve, and the patient died one month later. Necropsy not obtained.

Case 2.—S. S. (No. 250119), male, age 63. (Previously cited.[7])

Carcinoma of cystic duct with invasion of gallbladder, hepatic ducts and upper common bile duct. Died 22 days after operation. Necropsy revealed that hepatic artery had been resected with the tumor; there were infarcts in the liver.

Case 3.—G. C. (No. 285731), male, age 59.

Carcinoma of extrahepatic bile ducts with invasion of the gallbladder. Died six days after operation. During operation portal vein had been opened and ligation was necessary; a segment of hepatic artery was resected with the tumor. Necropsy revealed extensive septic infarction of the liver.

Case 4.—S. B. (No. 344040), female, age 56.

Carcinoma of extrahepatic bile ducts with invasion of the gallbladder and metastatic nodule in liver adjacent to the gallbladder. Living five months after excision. Icterus completely cleared. Moderately comfortable, but gradually losing weight.

Case 5.—M. M. (No. 321201), female, age 60.

Carcinoma extrahepatic bile ducts with involvement of gallbladder. Survived radical excision three months. Icterus reduced, but not relieved. Necropsy not obtained.

Case 6.—S. C. (No. 326441), female, age 72.

Carcinoma extrahepatic bile ducts with involvement of the gallbladder. Survived radical excision for five months. Icterus completely relieved. Moderate comfort during period of survival. Necropsy not obtained.

Case 7.—J. N. (No. 348567), female, age 49.

Carcinoma of lower common hepatic duct and upper common bile duct, with invasion of the gallbladder. Died 13 days after operation. Necropsy revealed bile peritonitis.

In general, the procedure, a choleangiocholecystocholedochectomy (Fig. 1), was as follows: Continuous spinal anesthesia supplemented by ethylene and ether if necessary.

1. High midline or reverse-L incision.
2. Aspiration of gallbladder if necessary to facilitate access to porta hepatis.
3. Dissection of the gallbladder from the liver bed and if extensively involved by carcinoma, wedge-shaped portion of liver excised with gallbladder,

cutting through the liver about .5 to 1 cm. wide of attached gallbladder. Hemorrhage from liver is controlled by large mattress sutures.

4. Application of hemostats to gallbladder and traction upon it to elevate mass in porta hepatis.

5. Isolation of lower segment of common duct behind duodenum after mobilization of duodenum and head of pancreas by incision of parietal peritoneum along the greater curvature of the duodenum.

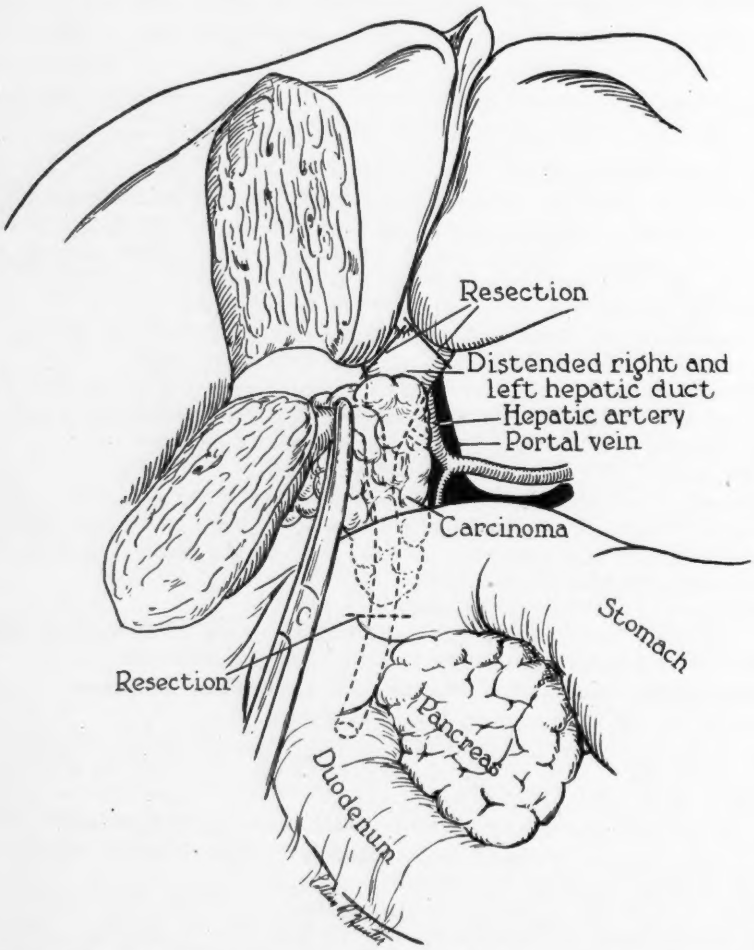

FIG. 1.—Schematic representation of resection of extrahepatic bile ducts, gallbladder and adjacent liver, for carcinoma involving these structures (Choleangiocholecystocholedochectomy).

6. Transection of common bile duct behind duodenum, and incisions into head of pancreas to mobilize portions invaded by carcinoma, if latter was present.

7. With hemostat applied to upper segment of transected common bile duct, dissection is carried out upward to free involved extrahepatic bile ducts from surrounding areolar tissue. This is the most precarious stage of the

operation since because of neoplastic invasion of areolar tissues from the ducts, the hepatic artery, and portal vein may be opened.

8. When carcinomatous ducts and attached gallbladder are freed except for right and left hepatic ducts, the latter are transected at or just beyond their emergence from the liver, and the specimen removed.

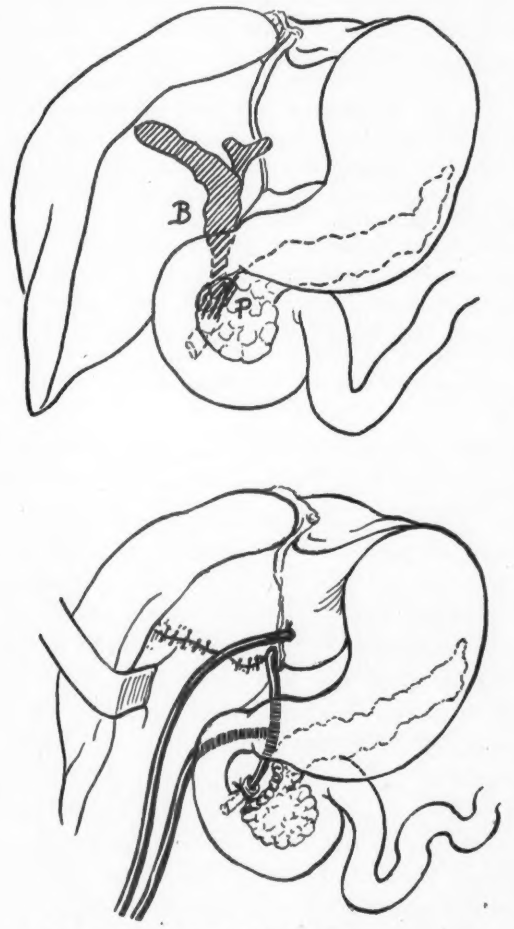

Fig. 2.—Case I S. P. (264586). A. Findings at operation—carcinoma (shaded area) involving practically entire extrahepatic biliary tract with invasion of head of pancreas. B. Termination of operation after excision of extrahepatic biliary tract, gallbladder, liver immediately surrounding it and portion of head of pancreas. T-Tube in right hepatic duct sinus and stump of common duct; urethral catheter in left hepatic duct stump. Survival 1 yr. (See text).

9. In the cases cited in this report the duodenum could not be mobilized to reach the liver, therefore, one of three procedures was carried out: (a) Two T-tubes were inserted, one in the right and one in the left hepatic duct stumps, and the lower arms inserted together in the lower stump of the common duct. (b) A T-tube was inserted between the right hepatic duct

and common duct stump, and an urethral catheter inserted in the left hepatic duct. (c) The left hepatic duct ligated, T-tube inserted between the right hepatic and common bile ducts. Soft rubber drains were placed in the right kidney fossa and the abdominal wound closed.

SUBSEQUENT COURSE IN PATIENTS SURVIVING OPERATION

Case 1.—(Fig. 2) T-tube between the right hepatic duct sinus in liver, and common bile duct stump near its termination; urethral catheter in left hepatic duct sinus. Urethral catheter came away two months after the operation. Wound healed about portion of T-tube, passing out of celiotomy incision with no discharge of bile about the tube. The latter was clamped and cut off just above skin level and left in place.

Case 4.—Left hepatic duct was ligated and T-tube placed between the right hepatic duct stump and stump of common bile duct. This tube came out two months after operation and was not replaced. Bile continues to be discharged from wound and patient receives bile salts by mouth. Icterus cleared.

Case 5.—Tube inserted as in Case 4. Copious biliary discharge about T-tube during three months period of survival.

Case 6.—Urethral catheters inserted in each hepatic duct stump. Practically the entire common duct was resected and lower segment ligated. Relief of icterus was the principal objective envisaged. Catheters came away two months after operation, and during remaining three months bile was discharged to the exterior. The patient received bile salts by mouth.

Injury to the Large Vessels in the Porta Hepatis as Complications of the Operation.—Injury to the portal vein and hepatic artery constitute the greatest hazards of the procedure. In Case 1, examination of the resected specimen revealed what was interpreted as a segment of hepatic artery constricted as a result of tumor growth about it. In Case 2, a segment of the hepatic artery was also resected, but this proved fatal in 22 days. In Case 3, both the portal vein and hepatic artery were opened and of necessity were clamped, the hemostats left in place for four days protruding from the abdominal wound and then removed. No hemorrhage ensued. At necropsy two days later, the occlusion of the portal vein and hepatic artery were confirmed. It is indeed surprising that survival was possible for six days with these vessels occluded. In Case 5 the right branch of the portal vein near the liver was accidentally opened as the resection was completed. Repair by suture was attempted but failed. Three Kocher hemostats were applied, left protruding from the wound and removed three days later without ensuing hemorrhage.

TABLE II

RECAPITULATION OF RESULTS

Survived operation: 4 patients
Operative mortality: 3 patients (43%)
(average survival 13 days)
Of those surviving:
One lived 1 year—icterus cleared, several months of return to normal activities.
One lived 5 months—icterus cleared. Palliation.
One lived 3 months—icterus partially relieved, and pruritus completely relieved.
One living 5 months—icterus cleared but general condition deteriorating.

Symptomatology.—Carcinoma arising in the extrahepatic bile ducts may infiltrate along the walls of these ducts and not form localized masses which

produce biliary obstruction early in the evolution of the process. Thus, an advanced stage of the disease is attained before icterus—a cardinal sign—obtains. The principal symptomatology: icterus, pain, "dyspepsia" and marked loss in weight exhibited by the patients reported above are summarized in Table III.

TABLE III

DURATION OF SYMPTOMS

Patient No.	Age	Icterus	Abdominal Pain	"Dyspepsia"	Loss of Weight
1.	67	2 wks.	0	0	0
2.	63	3 wks.	? 3 wks.	3 wks.	0
3.	59	6 mos.	0	9 mos.	40 lbs.— 9 mos.
4.	56	6 mos.	6 mos.	6 mos.	70 lbs.— 8 mos.
5.	60	7 wks.	1 yr.	?	30 lbs.— 3 mos.
6.	69	3 wks.	1 yr.	?	30 lbs.—10 mos.
7.	49	6 mos.	0	0	40 lbs.— 6 mos.

As stated, all of these patients presented advanced stages of carcinoma of the bile ducts, yet in four of the seven *icterus had been present for only two to seven weeks.* In Case 1, who had perhaps more extensive local involvement than the others, the icterus was of the briefest duration (two weeks). The abdominal pain when present was not characteristic, neither, of course, were the "dyspeptic" symptoms. Loss in weight was appreciable varying from 30 to 70 pounds over a period of three to nine months. Possibly greater attention to unaccountable loss in weight with otherwise negative findings on physical and roentgenologic examination might lead to exploratory celiotomy earlier in the course of this type of neoplasm.

Discussion.—The results achieved by radical excision in seven patients with advanced carcinoma of the extrahepatic biliary ducts do not permit of alteration in the very pessimistic outlook concerning this situation. Surgical attack, as radical as conditions permitted, was carried out and survival probably not appreciably lengthened in those recovering from the operation, although icterus was ameliorated. The one exception, Case 1 (age 67) did receive appreciable palliation, living for one year, most of the time free from icterus, and well enough to return to normal full-time occupation for a few months. Possibly a greater number of patients subjected to a radical operation might afford additional instances of satisfactory palliation. Furthermore, since the possibility of a radical procedure in this region is demonstrated, its performance in the presence of more localized carcinomas might increase the opportunity for more prolonged survival in such patients. On the other hand, the frequency with which icterus may develop very late in the course of carcinoma of the extrahepatic bile ducts adds greatly to the difficulties of diagnosis in an early stage. These results are reported in the spirit expressed by Sir James Walton[8] who stated that a "service a surgeon can make to the progress of the art and science of surgery is to discuss the difficulties he has met with in his practice." Furthermore, they represent what appears to be the limit of operative attack in one form of advanced abdominal cancer and what might be expected in the way of results.

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ARTERIOVENOUS ANEURYSM*

EXPOSURE OF THE TIBIAL AND PERONEAL VESSELS BY RESECTION OF THE FIBULA

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A near disaster from hemorrhage of the posterior tibial vessels in the course of the excision of an arteriovenous aneurysm has prompted the approach to these vessels by the removal of the upper portion of the fibula, including the resection of the head of that bone where necessary. The rich collateral anastomosis which develops as the result of an arteriovenous communication, together with dilatation of the vessels including those which perforate the interosseous membrane, demands direct visualization of these vessels and their careful ligation and division. Otherwise the retraction of vascular channels through the interosseous membrane may result in serious or even uncontrollable hemorrhage and necessitate a second incision along the front of the leg, or the removal of the fibula in the presence of hemorrhage and at an inopportune time during the course of the operation.

While this operation has been performed primarily as an approach to arteriovenous fistulae and aneurysms of the posterior tibial vessels, it is of equal importance to realize that the same approach is necessary to reach the anterior tibial and peroneal vessels in the upper part of their course. It is often impossible to differentiate by clinical measures which of these three vessels is involved because of their close proximity to each other. No matter which vessels are involved, difficulties in exposure are similar. In fact, resection of the upper end of the fibula is probably of more importance in fistulae involving the anterior tibial and peroneal vessels near their point of origin than it is of the posterior tibial. Moreover, more than one fistula may be encountered, as was found in Case 13.

The exposure of the fibula and the method of its removal have been described by Henry,[1] and it is but a modification of the procedure used by him which we have followed successfully in 15 instances. The fibula is removed subperiosteally, thus, insuring continued stability of the knee joint. Moreover, the peroneal nerve which may be concomitantly injured along with the vessels is at the same time exposed and may be explored or repaired without further operative incision.

It should be stressed, however, that resection of the fibula for exposure of these vessels is necessary only in their course in the upper portion of the leg. In the lower third they are more easily reached by direct approach along the posterior surface of the tibia on the medial side of the leg.

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* This article was to have been presented before the Annual Meeting of the American Surgical Association, May, 1945.

TECHNIC OF OPERATIVE PROCEDURE

Continuous spinal is the anesthetic of choice. A pneumatic tourniquet is applied to the thigh but is not inflated unless severe hemorrhage is encountered. The patient is placed upon the unaffected side, with the knee slightly flexed. Incision is carried directly over the fibula beginning about two inches above the head and extending distally for a length required by the position of the aneurysm (Fig. 1B). After the skin and superficial fascia are divided at the upper end of the incision, the deep fascia is opened at the medial edge of the biceps tendon. The common peroneal nerve is exposed, and a rubber strip passed around it for aid in mobilization (Fig. 1). The division of the deep fascia is carried downward along the course of the nerve along the posterior margin of the biceps tendon. The fascial origin of the peroneus longus muscle lies directly over the groove in which the nerve passes forward across the neck of the fibula. This fascia is divided. A definite plane, the lateral intermuscular septum, between the soleus muscle posteriorly and the peroneus longus muscle anteriorly, is easily developed and, when the muscles are separated, the lateral border of the fibula is immediately exposed. By the use of sharp dissection and a periosteal elevator the periosteum can be readily stripped from the fibula and its division accomplished by means of a Gigli saw (Fig. 2A). The subperiosteal removal of the head is more difficult and is best carried out by sharp knife dissection, keeping the blade of the knife *directly* against the bone and retracting the peroneal nerve completely out of the field of incision (Fig. 1A). With removal of the head and upper portion of the fibula, the lower end of the popliteal artery with its terminal branches, namely, posterior tibial, anterior tibial, and peroneal vessels, are exposed. With the retraction of the soleus muscle posteriorly and the peroneus longus muscle anteriorly, the vessels are easily seen (Figs. 3 and 4). The resected portion of the bone is not replaced.

In excision of the fistula, the artery proximal to it is secured as the first step in the procedure. A ligature is passed around it (for safety should severe bleeding be encountered) but not tied at this time. The vessels distal to the fistula are then isolated, ligated, and divided. The proximal artery is then ligated and divided. The fistula is then removed from below upward, ligating and dividing all communicating vessels. The proximal vein is divided as the last step in the operation. *It is of utmost importance that the region of the fistula be avoided until its principal blood supply is completely controlled.*

Following removal of the insertion of the fibular collateral ligament, the question of stability of the knee joint naturally arises. Subjectively, no patient has had complaint referable to the knee joint on the operated side. Examination has failed to reveal any loss of stability. Comparison of the fibular collateral ligaments by palpation, with the ligament under stress, usually discloses as tense a ligament as on the unoperated side.

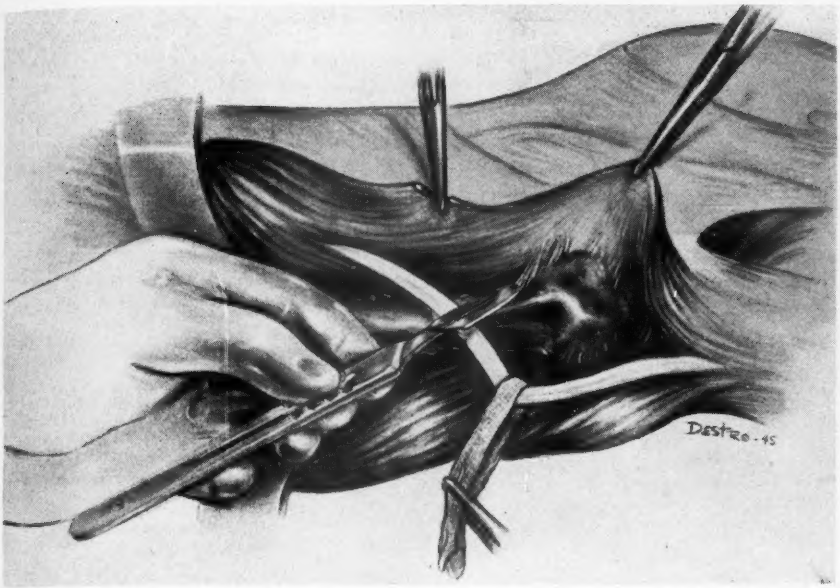


FIG. 1A.—Showing mobilization of peroneal nerve and sharp dissection of the periosteum covering head of fibula.

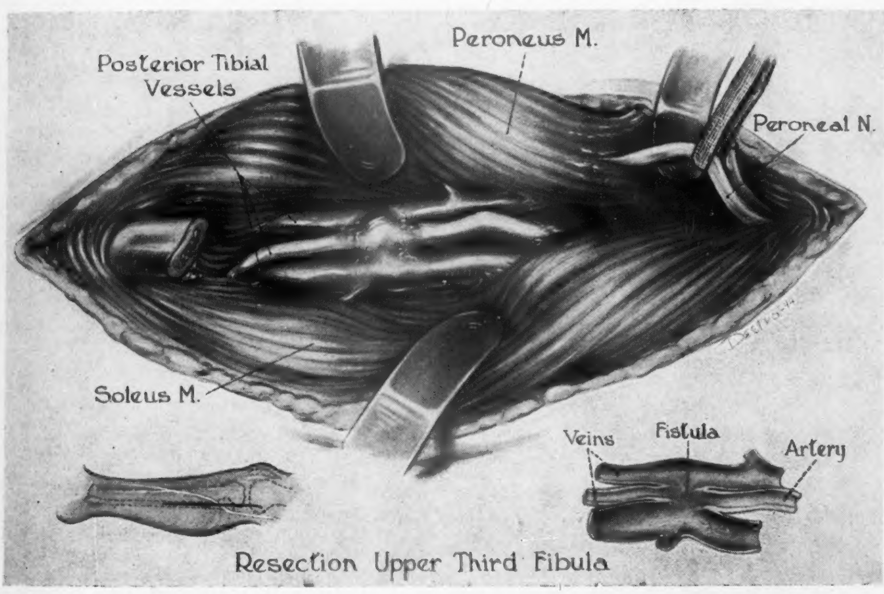


FIG. 1B.—The upper third of fibula removed, exposing fistula. Insert shows line of incision.

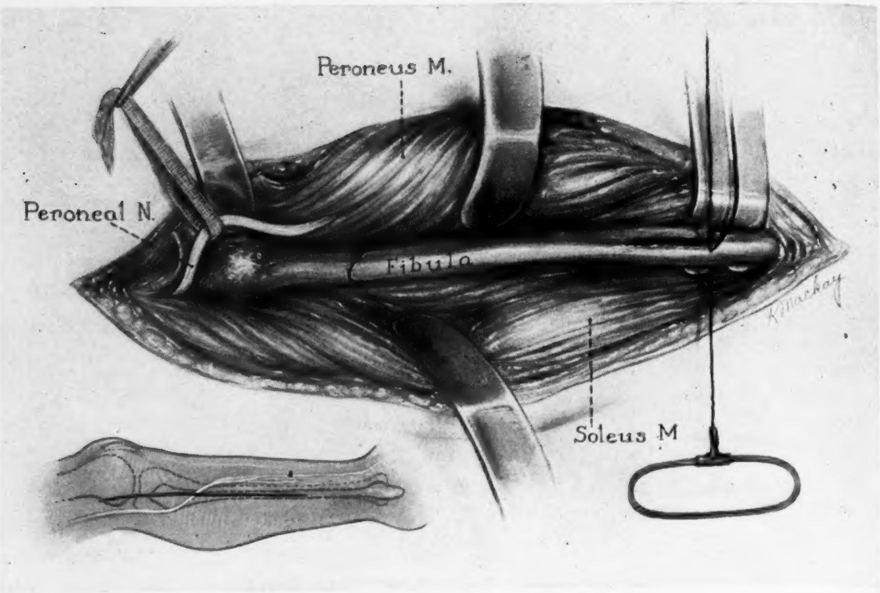


FIG. 2A.—Resection of fibula below the head.

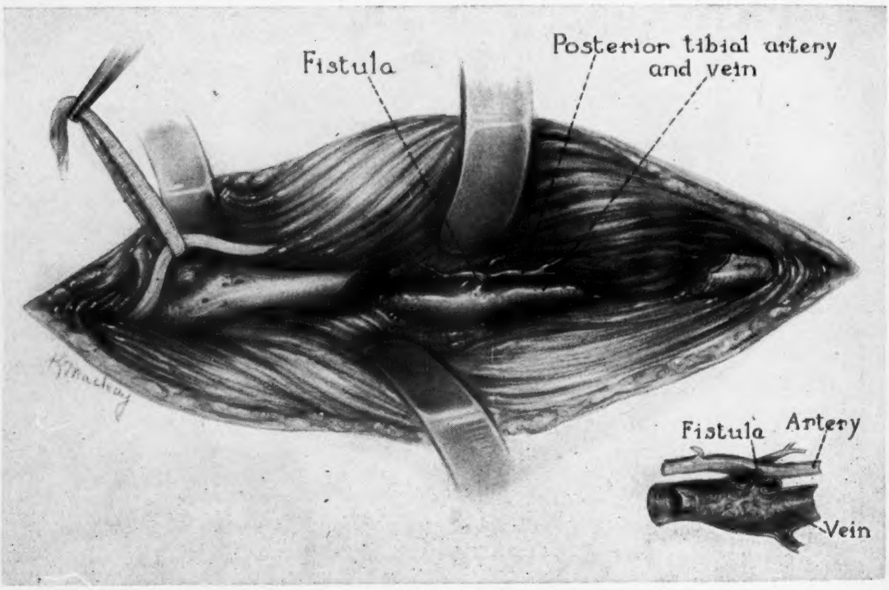


FIG. 2B.—Exposure of fistula between posterior tibial vessels.

CASE REPORTS

Case 1.—*A-V fistula, right posterior tibial vessels resulting from high explosive shell wound October 14, 1943. Resection of upper six inches of fibula not including the head. Quadruple ligation and excision of fistula, June 6, 1944. Recovery.*

On October 14, 1943, this 20-year-old soldier was wounded in the anterior middle portion of the right leg by high explosive shell fragment. There was little bleeding. On the same day the wound was débrided and immobilized by plaster. He was admitted to Ashford General Hospital February 14, 1944.

He complained of blueness and swelling of his right leg and foot on standing.

On examination, there was an infected wound on the anterior surface of the right leg. On elevation it became paler than the left. There was a well defined thrill and bruit, both anteriorly and posteriorly, at the junction of the upper and middle third of the leg. The bruit, which was continuous, was transmitted throughout the leg, into the foot, and upward to the middle of the thigh along the course of the vessels. Oscillations were markedly decreased at the right ankle and foot. On obliteration of the fistula by pressure the pulse rate dropped from 92 to 84 (Branham's sign). Radiography revealed no cardiac enlargement.

Drainage from the wound had ceased by May 1, 1944, and on June 6, 1944, operation was carried out. The six inches of fibula distal to the neck was resected and the fistula excised. The patient's recovery was uneventful, and there was no instability of the knee.

Case 2.—*A-V fistula, left posterior tibial vessels resulting from high explosive shell fragment September 17, 1943. Subperiosteal resection, upper half of fibula and excision of fistula June 28, 1944. Recovery.*

On September 17, 1944, this soldier sustained a shell fragment wound in the upper portion of the left leg, the missile entering anteriorly just below the knee. There was no wound of exit. Profuse bleeding necessitated control by tourniquet. Soon after the injury a drop foot was noted. The wound healed and the drop foot had disappeared by February 1, 1944. On March 9, 1944, at an overseas hospital, an attempted excision of the fistula was unsuccessful because the interosseous membrane interfered with proper ligation of the distal vessels. The operative note (Major John D. Martin, 43rd General Hospital) expressed the opinion that only by resection of the fibula could these vessels be secured. The proximal vessels were ligated at this time, but the bruit and thrill returned shortly thereafter, and the patient was evacuated to the Zone of Interior.

On admission to Ashford General Hospital he complained of pain in the left leg and foot, particularly after walking, and of swelling in the left leg.

There was a healed wound on the anterolateral aspect of the left leg in the upper third and a healed operative scar over the popliteal space. The left leg was considerably larger in circumference than the right. On dependency, the right toes and foot were cyanotic and on elevation there was a pallor of the left foot. The pulsation of the left dorsalis pedis artery was faint and that of the posterior tibial was absent. There was a thrill over the lower popliteal space and the upper anterior aspect of the leg and a continuous bruit, accentuated in systole, was transmitted throughout the leg and thigh. Obliteration of the bruit by pressure resulted in a diminution of the pulse rate from 72 to 60. Oscillometric readings at both popliteal levels were normal and equal. Oscillations at the left ankle were diminished and were absent in the left foot. Skin temperatures were increased over the leg in the region of the fistula and decreased at the toes. The cardiac diameter was normal.

On June 28, 1944, the upper half of the fibula, including the head, was resected subperiosteally. The fistula was easily identified because of the presence of large dilated veins. After securing the proximal and distal arteries, three large veins distal to the fistula were ligated and divided, as were numerous small communicating vessels. The fistula was removed in its entirety by ligating the vessels proximal to it as the last step in the procedure. Recovery was uneventful, and there was no instability of the knee. The patient returned to full duty as a paratrooper.

Case 3.—*A-V fistula, right posterior tibial vessels, caused by high explosive shell fragment February 19, 1944. Paralysis of the right superficial peroneal nerve. Subperiosteal excision of five inches of fibula distal to the neck. Excision of fistula. Exploration of peroneal nerve July 28, 1944. Cure of fistula. No improvement in nerve function.*

Following multiple shell fragment wounds, five of which involved the right leg and thigh, sustained February 19, 1944, this soldier suffered shock for which he was treated with plasma. After evacuation to the Zone of Interior some two and a half months after injury, he discovered a "buzzing" sensation in the region of the right knee, and was admitted to Ashford General Hospital July 19, 1944.

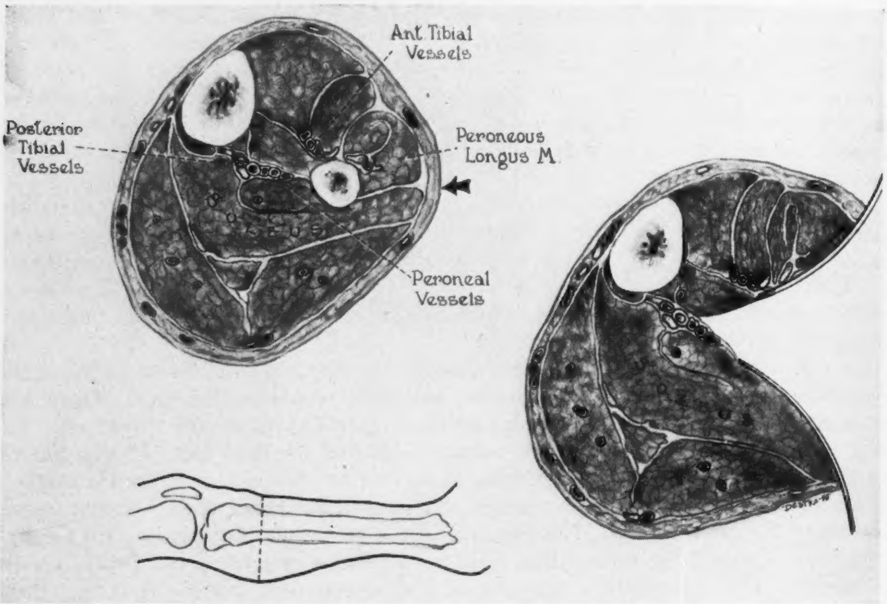


Fig. 3.—Cross-section through leg at level indicated in insert. Arrow shows point of incision through lateral intermuscular septum, with exposure of vessels after resection of fibula and retraction of muscles.

He complained of constant aching pain along the lateral aspect of the right leg and foot with alternating periods of cold and warmth in the right lower extremity and of right drop foot with anesthesia over the anterior portion of the right leg and dorsum of the right foot.

There was a considerable degree of atrophy of the right leg. Multiple wounds were present about the lateral aspect of the right lower thigh, knee, and upper leg. A large scar overlay the region just anterior to the head of the right fibula. Right drop foot was present. The right lower extremity was cyanotic. Oscillometric readings were increased at the right popliteal level and diminished at the right foot. Oscillations were normal on the left. Skin temperatures were diminished in all toes, more markedly on the right. There was a thrill over the lower popliteal space where a continuous bruit, accentuated in systole and transmitted both proximally and distally, could be heard. A striking finding was the intensity with which the bruit was transmitted along the anterior surface of the upper portion of the leg, indicating communication of the anterior vessels with the fistula. On obliteration of the fistula there was a drop in the pulse rate from 88 to 64.

At operation, July 28, 1944, five inches of the fibula distal to the neck was resected subperiosteally. The transverse fascial origin of the soleus muscle was divided, thus exposing the posterior tibial vessels in their upper portion. At this point the fistula was

located. The artery proximal to the fistula was dilated, as was the vein. The distal artery was small, but the veins were enlarged. After ligation and division of the proximal and distal arteries, a marked pulsation in the region of the fistula indicated other vessels of considerable size were entering this lesion. In the excision of the fistula several vessels, one of large size, were found to enter it through the interosseous membrane. All vessels were ligated and divided and the fistula removed in one mass.

The superficial peroneal nerve was explored and was found to be imbedded in scar tissue and to be the seat of considerable fibrosis. Recovery was uneventful, and there was considerable improvement in the nutrition of the leg, but there was no return of function in the muscles supplied by the superficial peroneal nerve. Although the heart was apparently not increased in size, there was a diminution of one centimeter in the transverse diameter following operation. He was fitted with a drop-foot brace and discharged from the service.

Case 4.—*A-V fistula, upper peroneal vessels, resulting from 25-caliber bullet June 21, 1944. Subperiosteal resection upper third of fibula and excision of fistula October 17, 1944. Recovery.*

On June 21, 1944, this soldier was struck in the left upper leg by 25-caliber bullet. He suffered a compound fracture of the upper third of the fibula. There was profuse bleeding, controlled by pressure. The wound was later débrided and the leg immobilized in plaster. On removal of the plaster six weeks later the presence of an arteriovenous fistula was discovered by a physiotherapist while giving massage. He was evacuated by air from the Pacific area and admitted to Ashford General Hospital September 15, 1944.

On admission, the patient complained of coldness and sweating of the left foot, tingling in the toes, stiffness of the left ankle and a "buzzing" sensation along the lateral aspect of the left leg.

There was a small healed wound of entry on the medial aspect of the left leg at the level of the tibial tubercle and a wound of exit on the lateral side of the leg at the junction of the middle and upper thirds. A thrill could be felt at the junction of the upper and middle thirds of the leg both anteriorly and posteriorly. A loud, harsh continuous bruit was most marked at this area and was transmitted along the course of the anterior and posterior tibial vessels and up the femoral vessels to the groin. On obliteration of the fistula the pulse rate fell from 84 to 74, and the blood pressure changed from 120/84 to 130/94. Transverse cardiac measurements were normal. The left foot was cyanotic on dependency and pallid on elevation. The oscillometric readings at the left popliteal level were higher than on the right. The skin temperature of the toes was two degrees lower on the right than on the left.

On October 17, 1944, operation was performed. The upper third of the left fibula including the head was subperiosteally resected. A fistula communicating with a false sac, one inch in diameter, was completely excised after ligating the major proximal and distal vessels and numerous communicating vessels (Fig. 5). The patient's recovery was uneventful.

Case 5.—*A-V fistula, left posterior tibial vessels resulting from shell fragments July 6, 1944. Subperiosteal resection, upper third of left fibula. Quadruple ligation and excision of fistula October 18, 1944. Recovery.*

On July 6, 1944, this 23-year-old soldier received multiple wounds of both lower extremities with a compound fracture of the upper third of the right fibula produced by shell fragments. The right leg at the site of the fracture bled profusely, and was controlled by a tourniquet. The following day débridement was carried out. A large wound of the left popliteal space was treated by skin graft. A month after the injury the presence of a thrill and bruit was noted along the upper outer aspect of the left leg, and he was evacuated to the Zone of Interior, and admitted to Ashford General Hospital October 2, 1944.

He complained of a "buzzing" sensation in the left leg, sweating of the left foot, and swelling on dependency.

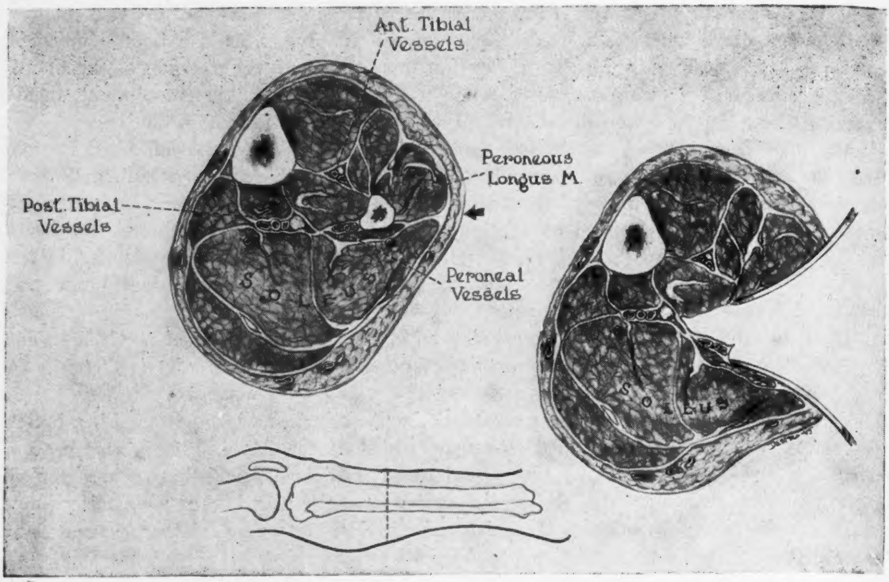


FIG. 4.—Cross-section of leg at level indicated in insert, showing exposure of vessels at this level after resection of fibula and retraction of muscles.

FIG. 5.—Case 4: Fistula with false sac. A window has been cut at the area of the fistula showing communication between artery and vein.

There were multiple wounds of both lower extremities and a large healed scar on the lateral upper aspect of the left leg and a well-healed area of grafted skin in the left popliteal space. There was a thrill along the upper lateral aspect of this leg with a rough continuous bruit heard loudest in the upper third of the leg and transmitted down the calf and upward to the groin. On obliteration of the fistula the pulse rate fell from 100 to 80, and the blood pressure changed from 120/90 to 140/100. Cardiac measurements were normal.

On October 18, 1944, the upper-third of the left fibula was resected subperiosteally, and after ligating numerous vessels of entrance and exit, a fistula between the upper tibial vessels was excised. Its position required ligation and division not only of the tibial vessels but of the origin of the anterior tibial and peroneal vessels as well. It was believed that successful ligation of these vessels could not have been carried out without resection of the fibula and subsequent easy access to the arteries and veins perforating the interosseous membrane. Recovery was uneventful.

Case 6.—*A-V fistula, left posterior tibial vessels resulting from shell fragments July 15, 1944. Complete paralysis peroneal nerve. Resection of upper third of fibula, excision of fistula, exploration of irreparable peroneal nerve injury, excision of scar, November 3, 1944. Mild secondary infection. Cure of A-V fistula. No improvement in nerve function.*

On July 1, 1944, this 22-year-old soldier was struck with shell fragments and suffered multiple wounds of the left lower extremity and hip. There was a severe wound overlying the upper half of the left fibula. None of the wounds bled excessively. The wounds were immediately débrided, shell fragments removed, and the wounds secondarily closed. Because of the peroneal nerve injury he was evacuated to the Zone of Interior and shortly thereafter evidence of an arteriovenous fistula of the left leg was noted. He was admitted to Ashford General Hospital October 18, 1944.

He complained of left drop foot, coldness, tingling, and numbness of the left leg and foot.

There were numerous healed sutured scars along the lateral and posterior aspects of the left lower extremity from the buttocks to the ankle. Just distal to the head of the left fibula there was a well defined thrill. A harsh continuous bruit, accentuated in systole, was best heard in this region and was transmitted upward to the groin and downward to the foot. Upon obliteration of the fistula by pressure the pulse rate fell from 80 to 60, and the blood pressure changed from 120/60 to 120/74. The cardiac measurements were normal. Oscillometric readings were increased at the left popliteal level and diminished in the left foot. The skin temperatures in the toes of both feet were below normal, and slightly lower in the left than on the right.

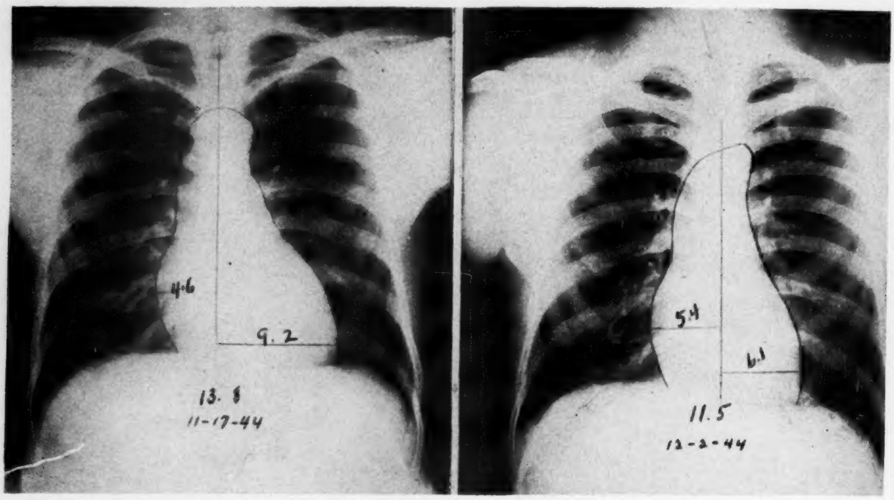
On November 3, 1944, the scar over the upper left fibula was excised, and the upper third of the fibula, including the head, was resected subperiosteally. A fistula between the posterior tibial vessels was found two centimeters distal to the origin of the peroneal artery. It was completely excised after ligating its major branches. A small false sac, lying on the lateral side of the fistula, was not removed. Following excision of the fistula exploration of the peroneal nerve was carried out. It was found to be divided at the point where it passed posterior to the head of the fibula. The distal portion was found in the substance of the peroneal muscles. Approximately four inches of this nerve had been shot away, and both ends were the seat of neuromas. After the removal of the neuromas the hiatus between the ends could not be breached, but the leg was flexed and a bridge of tantalum wire placed between the two ends in case further exploration was deemed advisable.

Following operation mild infection developed in the skin, probably the result of tension at the site of scar excision. The infection cleared rapidly without the necessity of secondary closure or skin graft. There has been some improvement in the nutrition of the leg following excision of the fistula. The extent of the nerve lesion probably precludes improvement in its function.

Case 7.—*A-V fistula, right anterior tibial vessels resulting from shell fragments, June 26, 1944. Resection of the upper third of the fibula, excision of the fistula November 25, 1944. Recovery.*

On June 22, 1944, this 25-year-old soldier received multiple shell wounds of both lower extremities, chest, eye, and nose. Although bleeding was profuse he was able to walk five miles to his own line. The wounds were débrided on June 25, 1944, and later were secondarily closed. About six weeks later a thrill in the upper right leg was discovered by a physiotherapist in the course of treatment. He was evacuated to the Zone of Interior, and admitted to Ashford General Hospital November 16, 1944.

He complained of pain in his right ankle, coldness, and excessive sweating of the right foot and a "buzzing" sensation along the upper anterior portion of the right leg.



A B

Fig. 6.—Case 7: A. Teleoroentgenogram of heart before operation. B. The heart shadow seven days after operation, showing reduction in cardiac diameter.

The right anterior thigh and leg were peppered with multiple wounds, well healed. There was a thrill over the anterolateral aspect of the upper portion of the right leg. It was also felt posteriorly in this region but not so marked as on the anterior surface. In this region a loud continuous bruit accentuated in systole was heard which was transmitted upward to the popliteal space and downward to the foot. On obliteration of the fistula the pulse rate fell from 72 to 52, and the blood pressure changed from 110/70 to 118/80.

On November 25, 1944, the upper third of the left fibula, including the head, was resected subperiosteally. The lower end of the popliteal artery was isolated and a suture, which was not tied, was passed around it. The fistula was found just distal to the origin of the anterior tibial artery, so near the popliteal artery that it was necessary to ligate that vessel in its removal. The anterior tibial artery and vein just distal to the fistula were ligated and divided and the fistula removed in its entirety. The transverse cardiac diameter was 13.8 cm. before operation. Two weeks after operation this had decreased to 11.5 cm. (Fig. 6). Recovery was uneventful.

Case 8.—*A-V fistula, right posterior tibial vessels, resulting from shell fragments, July 15, 1944. Resection upper third of fibula. Excision of fistula January 6, 1945. Recovery.*

On July 15, 1944, this 24-year-old soldier received multiple shell fragment wounds of the right leg, left foot, and both buttocks. Bleeding from the right leg was profuse,

but no tourniquet was applied. The injury was behind enemy lines, and he did not receive aid for ten hours. He was evacuated to England the following day, the wounds were débrided, and closed secondarily. Shortly thereafter the presence of an arteriovenous fistula was noted. The patient was evacuated to the Zone of Interior, and admitted to Ashford General Hospital on November 29, 1944.

He complained of "buzzing" sensation in the right upper leg with coldness and blueness of both feet, most marked on the right.

There were multiple small wounds over the anterior and posterior aspect of the right lower extremity. There was a long healed scar over the medial aspect of the right leg. There was a definite thrill over the anterolateral aspect in the right leg near the head of the fibula. A loud, harsh, continuous bruit could be heard over this region and was transmitted upward into the popliteal region and downward to the foot. Upon obliteration of the fistula the pulse rate fell from 72 to 64, and the blood pressure changed from 116/68 to 118/80.

On January 6, 1945, the upper third of the fibula, including the head, was resected subperiosteally and the posterior tibial vessels which were the seat of the fistula were exposed. The proximal artery and vein were identified and sutures passed around them which were not tied. The proximal vessels were greatly dilated. The veins distal to the fistula, which were dilated, were ligated and divided. The artery distal to the fistula, which was small, was ligated and divided. The proximal artery was then ligated and divided, and the fistula was removed from below upward after ligating numerous small vessels which entered it. The proximal vein was ligated as the last step in the procedure. The transverse diameter of the heart was 14.7 cm. before operation. Six weeks after operation this had decreased to 13 cm. Recovery was uneventful.

Case 9.—*A-V fistula, right posterior tibial vessels, resulting from shell fragments September 23, 1944. Resection of upper third of fibula. Excision of fistula January 12, 1945. Recovery.*

On September 23, 1944, due to mortar shell fragments, this 25-year-old soldier received multiple wounds of both lower extremities, chest, and right forearm. Bleeding was not profuse. Sixteen hours later the wounds were débrided, and at that time a compound fracture of the right tibia was found and the right leg was immobilized in plaster. Later, an arteriovenous fistula was found in the upper posterior surface of the right leg. He was evacuated to the Zone of Interior, and admitted to Ashford General Hospital December 19, 1944.

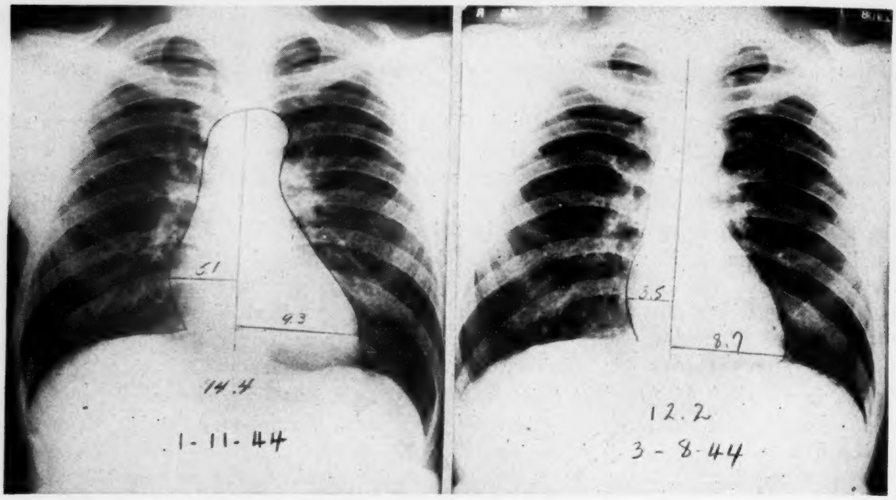
He complained of "buzzing" sensation in the upper part of the right leg, stiffness of the right knee, and coldness of the right leg.

There were numerous small, well-healed wounds over the leg, thigh, and body. On the upper posterior surface of the right leg there was a well-defined thrill and a continuous bruit transmitted upward to the popliteal vessels and downward to the foot. Upon obliteration of the fistula the pulse dropped from 96 to 80, and the blood pressure changed from 120/70 to 120/88.

On January 12, 1945, the upper third of the fibula, including the head, was removed. The fistula was located at the upper portion of the posterior tibial vessels. The proximal vein was dilated to twice its normal size. The artery, which was likewise dilated, was isolated and a suture passed around it, which was not tied. The tibial nerve was closely associated with the fistula and was dissected free from it. The distal veins were greatly dilated, and their ligation and division were accompanied with some difficulty because of hemorrhage resulting from the injury to one of them. This required mass ligation of the distal vessels. The proximal artery and vein were ligated and divided, and the aneurysmal mass was removed after ligating and dividing a number of communicating vessels. Prior to operation the transverse cardiac diameter was 14.4 cm. Two months after operation this had decreased to 12.2 cm. (Fig. 7). Recovery was uneventful.

Case 10.—*A-V fistula, right peroneal vessels secondary to machine gun wound September 4, 1944. Resection middle half of fibula. Quadruple ligation and excision of fistula, January 19, 1945. Recovery.*

This 32-year-old soldier was injured by machine gun fire on September 4, 1944, suffering a compound fracture of the right arm and wounds of both lower extremities. Shortly after the injuries the wounds were débrided and the right arm was placed in a plaster encasement. About a month after injury the patient discovered pulsation in the right calf and, because of this, he was evacuated to the Zone of Interior and eventually to Ashford General Hospital.



A B

FIG. 7.—Case 9:— A. Teleoroentgenogram of heart before operation. B. Reduction in cardiac diameter two months after operation.

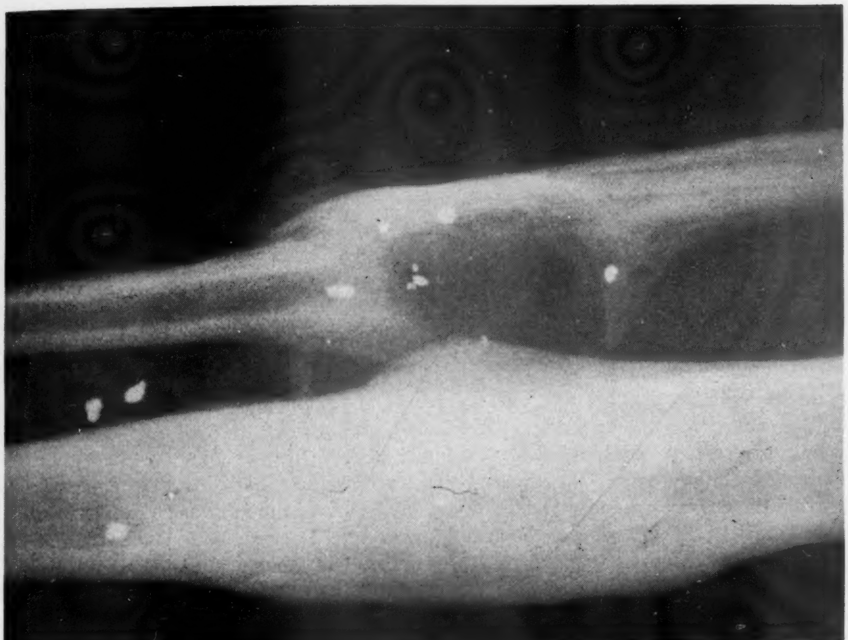
Physical examination showed incomplete union of the right humerus. The right leg, particularly the calf region, was swollen, and in the upper portion of this leg there was a well-defined thrill and continuous bruit, accentuated in systole. On obliteration of the bruit there was no drop in the pulse rate. The cardiac diameter was not increased.

On January 19, 1945, under spinal anesthesia, incision was made along the lateral border of the fibula and the middle half of that bone was excised. In separating the peroneus longus and soleus muscles the fistula was found in the peroneal vessels at the junction of the upper and middle thirds of the leg. It was completely excised after ligating its main branches and all communicating vessels. The excised fibula was not replaced. Patient's recovery was uneventful. He was transferred to the Orthopedic Section for continuation of treatment for the ununited fracture of the humerus.

Case 11.—*A-V fistula, with false sac, left peroneal vessels, resulting from shell fragments June 3, 1944. Resection middle third of fibula; excision of fistula with false sac January 26, 1945. Complete recovery.*

On June 3, 1944, this 21-year-old soldier was injured by mortar shell fragments which produced wounds of both legs and the right forearm. Bleeding from the left leg required a tourniquet. He was rapidly taken to an Evacuation Hospital where the wounds were débrided, and where a compound comminuted fracture of the left tibia and fibula was discovered. This was treated by plaster immobilization. In spite of some infection the wounds healed and immobilization was discontinued on September 6, 1944, at which time the patient became conscious of a pulsating "buzzing" mass in the left calf. This was later brought to the attention of his medical officer, and he was later brought to Ashford General Hospital on January 11, 1945.

Fig. 8



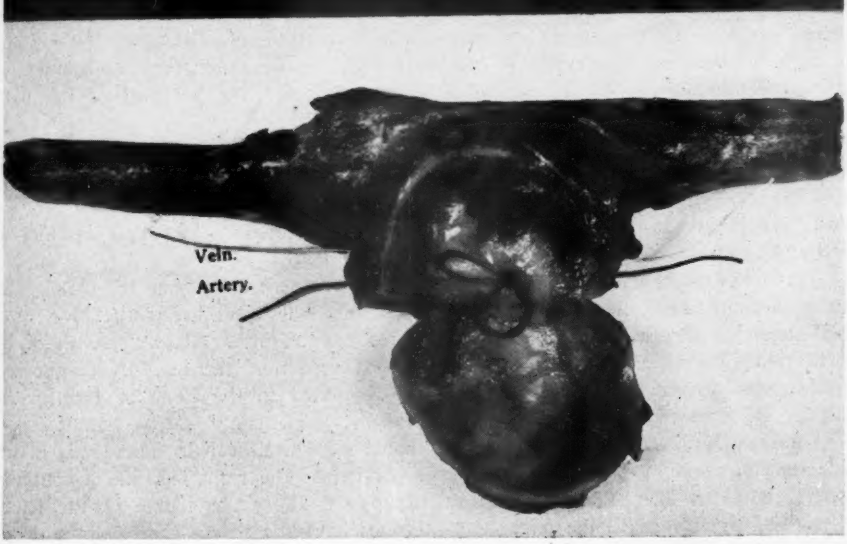


Fig. 9

Fig. 8.—Case 11: Roentgenogram showing pressure necrosis of fibula by aneurysm at site of fracture.

Fig. 9.—Case 11: The specimen, showing portion of fibula removed with false aneurysmal sac in eroded bone. The sac has been opened to show communication with artery and vein.

He complained of the pulsation in the left calf, aching of the left leg and ankle, with stiffness, swelling, and cyanosis of the foot. Radiography of the leg (Fig. 8) revealed a well-healed fracture of the left tibia and a healed fracture of the fibula, with considerable necrosis of the bone at the point of fracture, believed to be due to pressure necrosis of the aneurysm.

There were two wounds, six inches long, over the middle portion of the tibia and fibula which were well healed. In the midportion of the leg, just posterior to the fibula, there was a pulsating mass, about two inches in diameter, over which a continuous thrill could be felt. In this region there was a continuous bruit transmitted upward to the popliteal space and downward into the foot. Upon obliteration of the fistula the pulse rate dropped from 100 to 84; and the blood pressure changed from 130/70 to 120/82. The pulsation, which in this region was just posterior to the fibula at the point of fracture, was believed to be limited by the bone itself which formed a barrier to the aneurysm. A diagnosis of arteriovenous fistula of the posterior tibial vessels, with a communicating false sac eroding the posterior surface of the fibula and limited by that bone, was made. In view of these findings resection of the fibula as a preliminary to excision of the fistula and sac seemed imperative.

On January 26, 1945, the sac was partially emptied by elevation and application of a pressure bandage, and a pneumatic tourniquet about the thigh was inflated. The scar over the fibula was excised and approximately six inches of the middle third of the fibula was resected subperiosteally. Considerable difficulty was encountered because of the bizarre configuration of the fibula at this level resulting both from the healed fracture and from the erosive changes produced by the pressure of the false sac (Fig. 9). Following resection of the fibula the peroneal vessels were isolated, both proximal and distal to the sac. Although removal of the tourniquet had been contemplated at this point, some of the proximal veins, poorly demarcated in their collapsed condition, were torn. Because of the resultant venous bleeding, the source of which was difficult to determine, the remainder of the procedure was carried out with more than the usual difficulty because of lack of definition of the partially collapsed vessels. Following ligation of the main communicating vessels of the fistula and its excision, the tourniquet was removed and many small communicating vessels ligated. The removal of the fistula necessitated division of the neck of the false sac, which was removed separately. The transverse cardiac measurement before operation was 13.2 cm. Six weeks later this had decreased to 12.5 cm. Recovery was uneventful.

Case 12.—*A-V fistula, right posterior tibial vessels, resulting from shell fragment wound November 18, 1944. Resection upper third of fibula. Quadruple ligation and excision of fistula. Recovery.*

This 21-year-old soldier was wounded by shell fragment November 18, 1944. There was a through-and-through wound of the right upper leg just below the level of the tibial tubercle. Profuse bleeding was controlled by pressure. After a month of hospitalization swelling was noted along the medial aspect of the leg. A diagnosis of arteriovenous fistula was made, and he was evacuated to the Zone of Interior, and admitted to Ashford General Hospital February 7, 1945.

On examination, the wounds previously noted were well-healed. There was a thrill, both anteriorly and posteriorly in the upper portion of the right leg, and a continuous bruit accentuated in systole and transmitted upward along the course of the femoral vessels and downward to the foot. On obliteration of the fistula there was a drop in pulse rate from 80 to 64; and the blood pressure changed from 110/64 to 110/76. There was mild cyanosis on dependency of the right foot. The pulsations of the dorsalis pedis and posterior tibial vessels were diminished on the right side. Skin temperatures of the right toes were four degrees lower than on the left. The cardiac diameter was not increased.

On February 22, 1945, operation was performed and the upper half of the right fibula, including the head, was resected subperiosteally. The peroneal nerve was secured

and dissected free from surrounding tissues as a preliminary step in this operation. When the peroneal and soleus muscles were separated the posterior tibial vessels were brought into direct view. The fistula was found in the posterior tibial artery just proximal to the origin of the anterior tibial artery. It was completely excised by dividing the anterior tibial vessels as well as the posterior tibial vessels, both proximal and distal to the fistula. The fibula was not replaced. The deep fascia and skin were closed with interrupted sutures of silk. His recovery has been complete, and there is good stability of the knee joint.

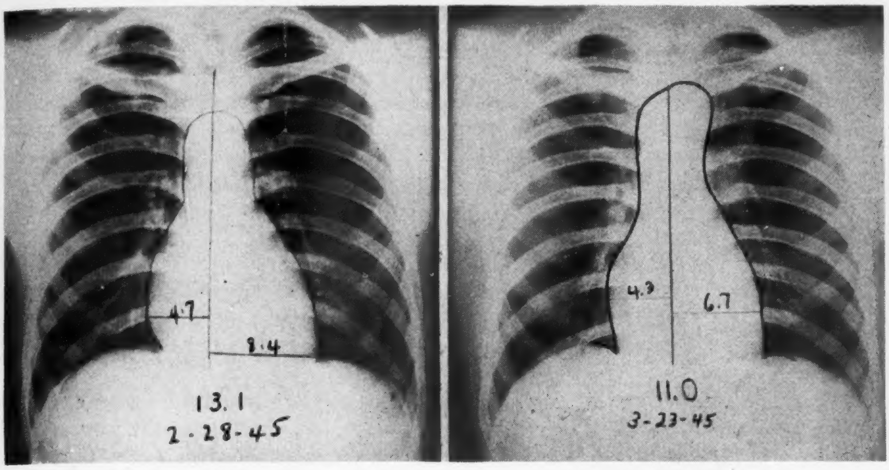


FIG. 10.—Case 14: A. Teleoroentgenogram of the heart before operation. B. The heart 15 days after operation, showing reduction in cardiac size.

Case 13.—*A-V aneurysm, with false sac, left peroneal vessels; A-V fistula anterior tibial vessels, both resulting from shell fragment wounds November 14, 1944. Resection upper third of fibula; proximal and distal ligation of peroneal vessels and intrasacular suture (Matas). Excision of A-V fistula anterior tibial vessels, March 1, 1945. Recovery.*

This 27-year-old soldier was wounded in action on November 14, 1944. He suffered one through-and-through wound in the upper portion of the left leg. A tourniquet was applied to control the bleeding. The wounds were débrided three days later. Two months after the injury the presence of an arteriovenous fistula was discovered in the left leg just below the knee, and for this he was evacuated to the Zone of Interior, and admitted to Ashford General Hospital February 15, 1945.

On examination, there were two well-healed wounds on the lateral and medial side of his leg about four inches below the knee. In the upper part of the calf there was an expansile pulsation with a continuous thrill. The thrill was transmitted all over the leg and up the thigh along the course of the femoral vessels. It was particularly prominent over the pulsating mass and on the anterior surface of the leg just below the knee. A continuous bruit accentuated in systole was heard over the leg and transmitted upward to the groin. Obliteration of the fistula by pressure caused a drop in pulse rate from 72 to 60; and a change in blood pressure from 110/60 to 110/80. There was marked increase in oscillometry in the region of the fistula.

On March 1, 1945, the upper half of the fibula, including the head, was resected subperiosteally. During the resection of the fibula considerable bleeding was encountered because the false sac was entered in the stripping of the periosteum from the bone. A tourniquet previously placed about the thigh was inflated. The fistula was found to be in the peroneal vessels which entered a large false sac. The proximal and distal

vessels were ligated and divided and the sac opened and five openings were closed by intrasacular suture, after the method of Matas. The tourniquet was removed and all bleeding points were ligated. It was then found that another fistula existed between the anterior tibial vessels about two inches distal to the origin of this artery. This fistula was excised after ligating the proximal and distal vessels which entered it. The resection of the fibula gave excellent exposure to both fistulae, and it was felt that this exposure could not have been obtained without a preliminary resection of the bone. His recovery was uneventful.

Case 14.—*A-V fistula, with false sac, left peroneal vessels, due to bullet wound sustained November 24, 1944. Resection of upper third of fibula, excision of fistula and sac, scar excision, and secondary closure with skin graft, March 8, 1945. Superficial infection. Recovery.*

This 21-year-old soldier was wounded by a bullet on November 24, 1944, the missile entering the medial aspect of the leg about three inches below the knee. A tourniquet was necessary to control hemorrhage. Shortly thereafter a compound fracture of the middle third of the fibula was débrided and the leg immobilized in plaster. Upon removal of the plaster the presence of an arteriovenous fistula was discovered, and he was admitted to Ashford General Hospital February 22, 1945.

Upon examination, there was a well-defined thrill and bruit over the lower popliteal space which was transmitted up the course of the femoral vessels and down into the leg. There were two longitudinal scars on the posterior aspect of the left leg. Upon obliteration of the fistula the pulse rate dropped from 72 to 60; and the blood pressure changed from 130/70 to 130/90.

Operation was performed March 8, 1945, and a ten-inch scar on the lateral surface of the leg was excised, and incision was carried three inches above the knee. After isolating the peroneal nerve, the upper third of the fibula, including the head, was resected subperiosteally. The fibula had previously been fractured at the junction of the proximal and middle thirds, and approximately one inch of the distal fragment was likewise resected. After the posterior periosteum was opened the fistula was found to be between the peroneal artery and vein, about four inches below the knee. The proximal and distal vessels were identified and ligated, and the fistula, together with the false sac, was completely excised. Several communicating vessels of considerable size were divided at the point where they entered the fistula. It was necessary to make a relaxing incision on the medial aspect of the leg in order to close the wound, and this was covered with a split-thickness skin graft. Some superficial infection developed in the wound, probably due to tightness of the closure. This cleared up rapidly. Prior to operation the transverse cardiac diameter was 13.1 cm., which decreased to 11 cm. ten days after operation (Fig. 10). The grafted area of skin healed normally, and there is no evidence of recurrence of the fistula.

Case 15.—*A-V fistula with false sac, left anterior tibial vessels, due to land mine fragments, November 28, 1944. Resection upper third of fibula, quadruple ligation and division anterior tibial vessels; intrasacular suture of false sac (Matas) March 15, 1945. Recovery.*

This 26-year-old soldier was wounded by fragments of a land mine on November 28, 1944. He suffered multiple wounds of both lower extremities, and a through-and-through wound of the upper portion of the left leg. Bleeding was not profuse from any wound. Shortly thereafter the wounds were débrided. The patient was conscious of a throbbing in the upper portion of the left leg soon after the injury, but it was not until after evacuation to the Zone of Interior that presence of a fistula was noted. He was admitted to Ashford General Hospital on March 1, 1945.

On examination, there were numerous small healed wounds in both lower extremities. There was a larger wound of entry on the upper portion of the calf three inches below the knee. There was a large pulsating mass on the anterior portion of the leg just below the knee and, in addition, a continuous thrill and bruit, accentuated in systole, heard all

over the upper portion of the leg and transmitted to the foot and to the groin. On obliteration of the fistula the pulse rate dropped from 76 to 64. There was no change in the blood pressure, which was 110/70. Transverse cardiac diameter was normal.

Operation was performed March 15, 1945. The upper half of the fibula, including the head, was resected subperiosteally. In removing the fibula it was found that this bone formed a portion of the false sac, which was opened. Bleeding was controlled by the inflation of a pneumatic tourniquet which had been applied previously. The sac was opened and dissection revealed that the sac communicated with the anterior tibial artery and vein. These vessels were isolated both proximal and distal to the sac, and ligated and divided. The openings in the sac were closed with interrupted sutures. A portion of the sac was excised, and the remainder was infolded with a series of sutures. On removal of the tourniquet there was no bleeding, and the wound was closed in the usual manner in layers. Recovery was uneventful.

SUMMARY AND CONCLUSIONS

Careful exposure of the tibial and peroneal vessels in the upper part of their course is necessary in the operative treatment of arteriovenous fistulae in this region. This is facilitated by subperiosteal resection of the fibula, including the head of the bone if necessary. The resected portion of bone is not replaced. There has been no instability of the knee joint following this operation. Fifteen consecutive cases in which this procedure was carried out are presented in abstract.

We are indebted to Kathleen Mackay and T/5 V. Destro for the illustrative drawings, and to Captain Floyd B. Hall and T/5 Joseph Jackson for photographs.

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THE SURGICAL TREATMENT OF THE MORE COMMON TYPES OF DIAPHRAGMATIC HERNIA: ESOPHAGEAL HIATUS, TRAUMATIC, PLEUROPERITONEAL HIATUS, CONGENITAL ABSENCE AND FORAMEN OF MORGAGNI*

REPORT OF 404 CASES

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ALTHOUGH THE OCCURRENCE of herniation of abdominal viscera through the diaphgram is relatively uncommon when compared with herniation through the abdominal wall, there are more different kinds of hernia occurring through the diaphragm than there are occurring through the other walls which encase the abdominal contents. The reason for the different types of diaphragmatic hernia is the unusual embryologic formation of the diaphragm, which makes it more susceptible to weak areas through which these herniae may occur.

The formation of the diaphragm from embryonic structures is a highly complex process, because the muscular elements of the diaphragm are derived from several sources. The anterior, lateral and central parts, which comprise the greater portion of the diaphragm in the adult person, are formed from the transverse septum and fused ventral mesentery. The remaining, posterolateral portion is formed by the fusion of the dorsal mesentery and the mesoderm derived from the receding wolffian body with the pleuroperitoneal membrane derived from the pulmonary ridge. It is difficult to determine the exact amount of the muscle tissue that is derived from each of these structures, since considerable variation probably occurs during the process, but it is likely that the dorsal mesentery forms the posterior and central portions, which contain the esophageal opening. The mesodermal cells from the receding wolffian body form the right and left crura. The pleuroperitoneal membrane grows ventrally, closes the remaining opening (hiatus pleuroperitonealis) between the peritoneal celom and the pleural celom by fusion with the transverse septum and forms the lateral portion of the diaphragm.

Failure of fusion or failure of proper deposition of the mesoderm at any one of these adjacent points of union may result in congenital continuity of the pleural and peritoneal cavities or a congenitally weak portion in the diaphragm at any of these points. Consequently, from an embryologic standpoint, weak portions might be expected to appear at the points of fusion of these different structures. These portions are situated dorsolaterally at the fissura pleuroperitonealis (foramen of Bochdalek) and also through the outer crus and through the esophageal opening. Herniation through the

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dome is common but cannot be explained on the foregoing basis, because the dome, embryologically, is not a fusion region. Such a hernia may be the result of excessive degeneration of the muscle in the formation of the central tendon or of some pathologic condition. Unilateral absence of the diaphragm probably is the result of the failure of development of the pleuroperitoneal membrane, which usually is found as a narrow ridge of tissue along the posterior wall of the thorax.

CLASSIFICATION OF DIAPHRAGMATIC HERNIA

There are numerous classifications of diaphragmatic hernia which are based on the embryologic and etiologic aspects, pathologic anatomy, the site of the opening in the diaphragm, the presence or absence of a sac, the contents of the hernia, and other factors. It is difficult, or impossible, to make most of these classifications clinically; accordingly, many of them are of little practical value.

All types of true hernia have a sac as one of the component parts; so that many conditions, commonly included under this term but in which there are no hernial sacs, would more properly be termed "evisceration" or "false hernia." The presence or absence of an hernial sac cannot be determined by clinical examination. It can be found only at operation.

From a clinical and surgical standpoint, the history of a preceding injury is helpful in establishment of the diagnosis and in determination of the type, urgency and prognosis of the operative treatment. Because of the practical clinical and surgical significance of trauma as an etiologic factor, I have suggested that diaphragmatic hernia be classified into two main groups: Nontraumatic and traumatic. I have subdivided these two groups according to the various types.

Nontraumatic Hernia.—A nontraumatic diaphragmatic hernia may be congenital or acquired. If it is congenital, the hernia is attributable to embryologic deficiency and usually does not have an hernial sac. The most common sites of a congenital hernia, in the probable order of frequency of occurrence, are: (1) Through the hiatus pleuroperitonealis (foramen of Bochdalek); (2) through the esophageal hiatus; (3) through an anterior substernal opening (foramen of Morgagni or Larrey's space); and (4) through the gap left by partial absence of the diaphragm, a gap which is usually situated in the posterior portion of the muscle.

If the hernia is acquired after birth, the sites of occurrence are: (1) Through the esophageal hiatus, a type in which there is an hernial sac; (2) through the region of fusion of the anlage of the diaphragm; and (3) at sites named under the congenital type in the foregoing paragraph.

Traumatic Hernia.—Traumatic diaphragmatic hernia may be caused by direct or indirect injury or by inflammatory necrosis of the diaphragm. In case of indirect injury of the diaphragm, the hernia may occur at any point, including points of embryologic fusion, but the most common sites are the dome and the posterior half of the left part of the diaphragm. On the other

hand, the hernia may occur in the right part of the diaphragm. It usually is the result of a severe, crushing injury. When the hernia occurs through the esophageal opening there is a sac but when it occurs through the leaf of the diaphragm there usually is no sac. In case of direct injury of the diaphragm, the hernia may occur at any point and is usually the result of penetrating wounds, such as those inflicted by a gun or knife.

Rupture of the diaphragm may be the result of inflammatory necrosis, which, in turn, has been caused by subdiaphragmatic abscess. Again, rupture may follow necrosis caused by drainage tubes which have been introduced into empyematic cavities. In these cases the opening usually is situated in the posterior part of the diaphragm and there is no hernial sac.

CLINICAL AND SURGICAL CONSIDERATIONS

In my experience, the most common types of diaphragmatic herniae, in order of frequency, which require surgical treatment are esophageal hiatus hernia; herniae due to trauma, indirect or direct, or to inflammatory necrosis; absence of a portion of the diaphragm; hiatus pleuroperitonealis herniae and herniae through the foramen of Morgagni (Fig. 1).

The number of diaphragmatic herniae of each of these types in the 404 cases in which I have operated is shown in Table I. Each of these various types of diaphragmatic hernia presents different clinical manifestations as well as different methods of surgical treatment. It will not be possible to go into detail but I shall present some of the more important clinical and surgical considerations of these different types of diaphragmatic hernia.

The clinical recognition of diaphragmatic hernia on the basis of the subjective symptoms alone is often very difficult. The symptoms are complex because of the various structures involved in the hernia and depend on the amount of mechanical interference with the function of the herniated abdominal viscera, on the degree of impairment of the normal function of the diaphragm and on the amount of increased pressure within the thorax which causes impairment of respiration and circulation.

The clinical syndrome of diaphragmatic hernia may be divided into two main groups. The first group occurs in cases in which the stomach is the only abdominal organ involved in the hernia. The symptoms are those of intermittent and usually progressive incarceration and obstruction of the stomach. The most common type of diaphragmatic hernia in which the stomach is the only abdominal viscus involved is through the esophageal hiatus. However, this type of hernia may contain various portions of the omentum, depending on the amount of stomach involved in the hernia. Inasmuch as these herniae are progressive, the entire stomach may become involved in the hernia and in these herniae the colon may also become incorporated in the hernial sac because of its attachment to the greater curvature of the stomach. More rarely the spleen may become involved because of its attachment to the cardia of the stomach. In these cases, in

TABLE I

DATA IN 404 CASES OF DIAPHRAGMATIC HERNIA IN WHICH OPERATION WAS PERFORMED

Site of Opening	Cases	Cause	Contents of Hernia	Cases
Esophageal hiatus	287	Congenital (history of trauma, 17)	Stomach (omentum)	266
			Stomach, omentum and spleen	6
			Stomach and colon	15
Short esophagus type	33	Congenital (11)	Stomach only	33
Hiatus pleuroperitonealis	9	Congenital	Right colon and small bowel	4
			Colon, small bowel, stomach and spleen	4
			Colon, small bowel and appendix	1
Absence of posterior fourth of diaphragm	12	Congenital	Stomach, colon, small bowel and spleen	5
			Small bowel and colon	2
			Small bowel, colon, spleen, appendix (3) and stomach (1)	5
Foramen of Morgagni (subcostosternal)	8	Congenital (right diaphragm 6; bilateral, 1)	Colon and omentum	7
			Stomach and colon	1
Traumatic: Left diaphragm	54	Trauma (indirect injury, 36; direct injury, 8)	Stomach only	7
			Stomach and colon	12
		Inflammatory necrosis (6)	Stomach, colon, small bowel (30), spleen (20) and liver (12)	35
Right diaphragm	1	Trauma (direct)	Stomach, colon, small bowel, liver (gallbladder) and head of pancreas	1
Total	404			404

TABLE II

SURGICAL PROCEDURES AND OPERATIVE RESULTS IN 404 CASES

Radical repair of defect in diaphragm:		
Approach: Abdominal, 369; thoracic, 2		371
Preliminary interruption of phrenic nerve	268	
Preliminary extrapleural thoracoplasty	3	
Operations in conjunction with repair of hernia:		
Gastric resection for gastric ulcer, 1; for carcinoma, 2	3	
Closure of perforated gastric erosion, 2 (Total erosions, 37)	2	
Gastro-enterostomy for gastric ulcer, 1; for duodenal ulcer, 2	3	
Splenectomy for tuberculosis, 2; for injury, 8	10	
Appendicostomy for obstruction	1	
Appendicectomy for appendicitis	2	
Interruption of left phrenic nerve (hiatus hernia):		
Palliative, 7; therapeutic, 26		33
Total patients operated upon		404

Recurrence of hernia of all types after operation:

Traumatic hernia, 0; congenital defect, 1; esophageal hiatus, 9

Recurrence of esophageal hiatus herniae, 9

Roentgenologic diagnosis, without recurrence of symptoms, 5

Roentgenologic diagnosis, with recurrence of symptoms, 4; repair of recurrent herniae, 4

Operative deaths, 16 or 4.0% (basis of 404 patients operated upon)

which the colon is involved, there may be additional symptoms of partial or complete intestinal obstruction.

The second group consists of those cases in which multiple abdominal viscera are involved in the hernia. These herniae are usually of traumatic origin and are caused by laceration of a normal diaphragm. However, they also may be of congenital origin and may result from congenital structural deficiency of the diaphragm. The symptoms in these cases are more varied and severe than those in the first group because of the multiple structures involved and are often more acute in onset. The initial symptoms may be those of acute intestinal or gastric obstruction or severe hemorrhage.

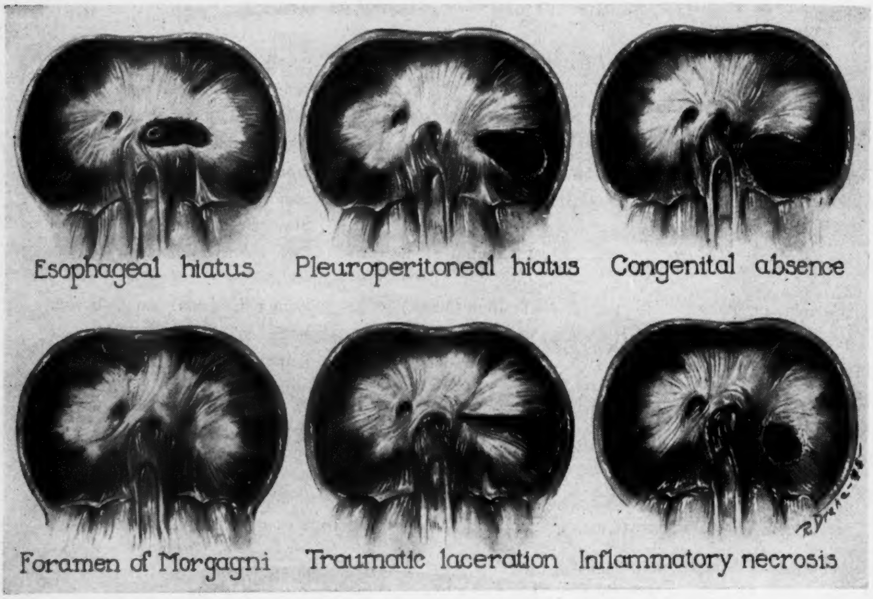

FIG. 1.—Situations of congenital structural defects and traumatic lacerations of the diaphragm which cause the more common types of diaphragmatic hernia.

From the standpoint of treatment, diaphragmatic hernia is primarily a mechanical condition and the only treatment which will relieve the condition is operative repair or reconstruction of the abnormal opening in the diaphragm after replacement of the herniated viscera into the abdomen. The indications for surgical intervention and methods and technic of surgical procedures depend on the type, situation and size of the defect in the structure of the diaphragmatic muscle, the kind and amount of abdominal viscera involved in the hernia and whether or not the viscera are enclosed in the hernial sac.

In the treatment of all herniae that have occurred through the left portion of the diaphragm, I prefer the abdominal approach by means of an oblique left rectus incision, starting at the ensiform cartilage and extending to the outer border of the rectus muscle. I believe there is less risk of the occurrence of thoracic complications when this approach is used. It is of particular advantage in cases of esophageal hernia, for the herniated stomach is usually

confined in a sac in the posterior part of the mediastinum and does not enter the true pleural cavity.

In the repair of herniae through the right portion of the diaphragm, I prefer the thoracic approach because the large, right lobe of the liver makes the abnormal opening in the diaphragm inaccessible from the abdominal approach.

The technical difficulties of adequate exposure of the hernial openings through the left portion of the diaphragm and the esophageal hiatus are often considerable because of fixation of the left lobe of the liver to the leaf of the diaphragm. The exposure of these hernial openings is greatly facilitated by cutting the suspensory ligament and retracting the left lobe of the liver to the right. This can be accomplished, when the left lobe is small, by folding it on itself, and when it is large, by retracting it forward into the wound. The spleen is often very adherent to the posterior part of the diaphragm and hernial openings, but usually can be separated from these structures by blunt dissection. In some instances the spleen has been so traumatized by the injury, and so bound into its abnormal position by adhesions, that it cannot be separated from the hernial opening without seriously injuring it. This not uncommonly occurs in the traumatic types of hernia, and occasionally in esophageal hiatus hernia. In these cases splenectomy is necessary.

Paralysis of the diaphragm, produced by temporary or permanent interruption of the phrenic nerve, is of value as a procedure preliminary to radical operative repair of esophageal hiatus herniae. It is a necessary procedure in the surgical treatment of partial thoracic stomach resulting from a congenitally short esophagus. In some cases in which radical operative repair is contraindicated, it may be used as a palliative measure.

ESOPHAGEAL HIATUS HERNIA

Herniation of the abdominal viscera through the esophageal hiatus is the most common type of diaphragmatic hernia occurring in adult life. These herniae are of considerable general interest because of the relative frequency of their occurrence, their indefinite causation, the variation of the relationship between the defective esophageal hiatus and the structures involved in the hernia, the progressive character of their development, the varied and complex symptoms produced by them and because of their treatment, which may be conservative if the herniae are small and symptoms mild but which may embrace surgical treatment if the herniae are large.

The symptoms of esophageal hiatus hernia may begin at birth or at any time during later life. Because of the progressive character of this type of hernia, the symptoms vary as the hernia becomes larger, depending on the degree and type of herniation present. Therefore, several different clinical diagnoses can be made in the same case, depending on the time at which the patient is examined, because of the changing symptoms. Accordingly, the condition may be termed the "masquerader" of the upper part of the abdomen. This, I believe, is the most important clinical consideration of

esophageal hiatus diaphragmatic herniae. In a study of 320 cases of this type of hernia in which I have performed operations, it was found that an average of three previous erroneous clinical diagnoses had been made in these cases before the correct diagnosis was established. The most common

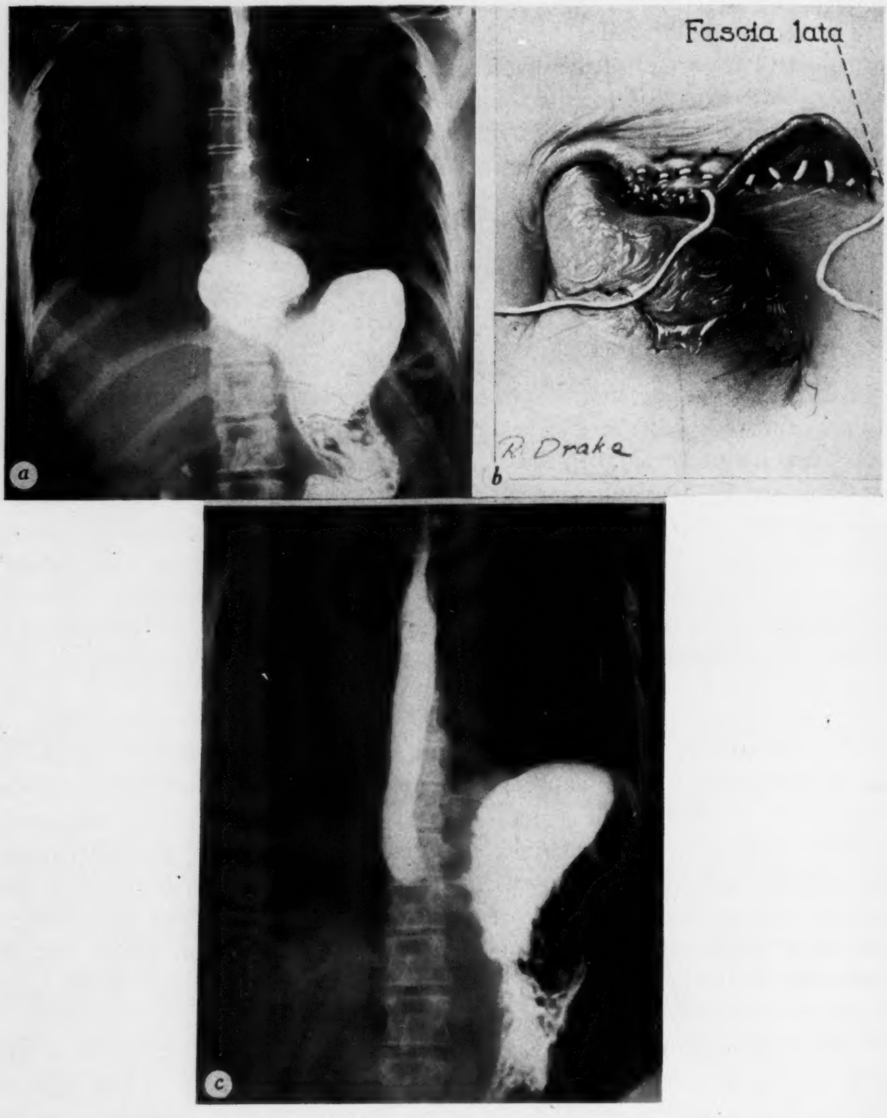

FIG. 2a.—Patient, age 49. Esophageal hiatus hernia with herniation of the cardiac end of the stomach through the esophageal hiatus and some displacement of the lower part of the esophagus (previously diagnosed gallbladder disease).

b. Same patient. Enlarged esophageal hiatus repaired to the left of the esophagus with interrupted silk sutures and continuous sutures of fascia lata by lapping the anterior over the posterior margin of the opening.

c. Same patient three weeks after repair of the hernia. The entire stomach is below the diaphragm, which is elevated because of temporary interruption of the phrenic nerve. The esophagus is in normal position.

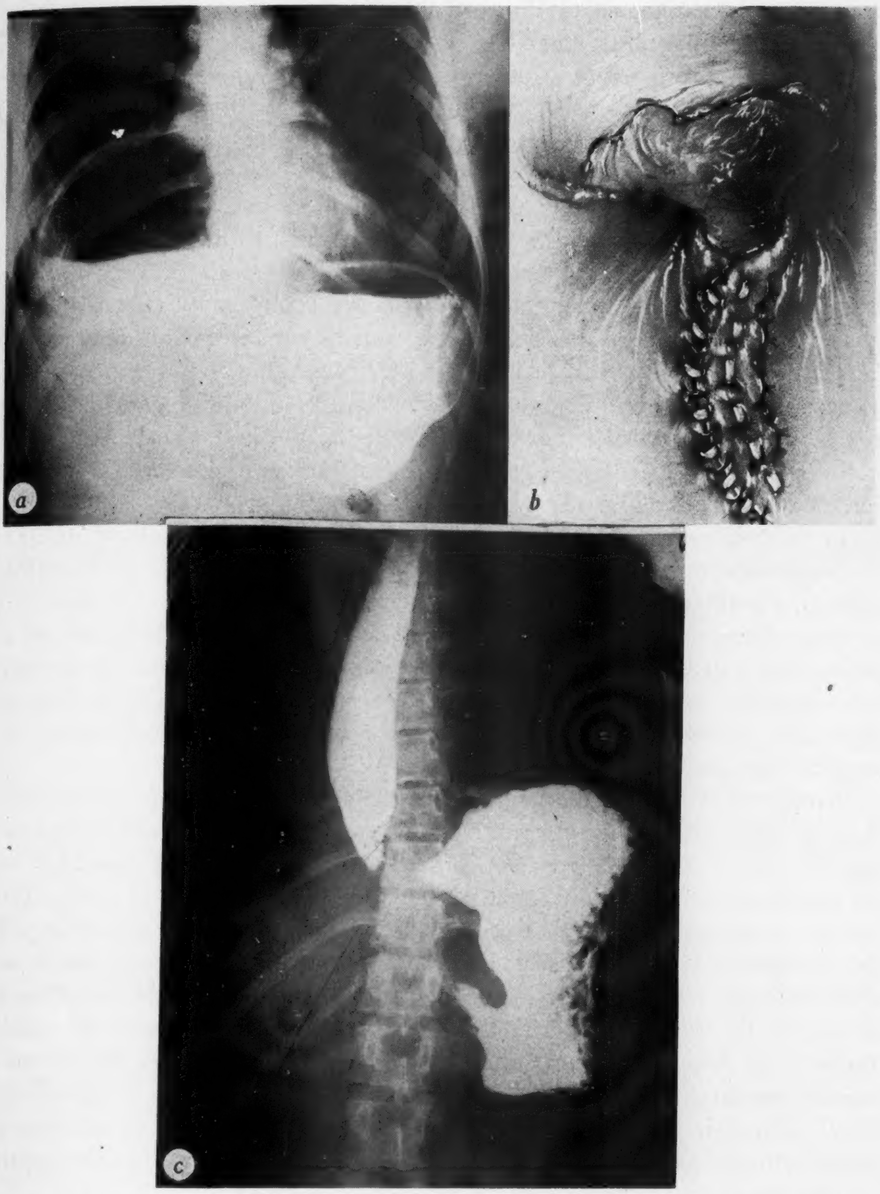

FIG. 3*a*.—Patient, age 17. Esophageal hiatus hernia with herniation of the pyloric two-thirds of the stomach into the right thoracic cavity. Stomach is enormously dilated due to incarceration and obstruction (previously diagnosed ulcer with obstruction).

b. Same patient. The enlarged hiatus, defective posteriorly, is repaired with interrupted silk sutures and continuous sutures of fascia lata by overlapping laterally in front of the aorta.

c. Same patient on dismissal, showing the entire stomach, in normal position below the diaphragm, which is elevated as result of temporary interruption of the phrenic nerve. The esophagus is in normal position.

erroneous diagnoses, in order of frequency, were found to be cholecystitis, cholelithiasis, gastric ulcer, duodenal ulcer, hyperacidity, secondary anemia, cardiac disease, carcinoma of the cardia, stricture of the esophagus, appendicitis and intestinal obstruction. In 32 of these cases the patients had been operated upon previously for other conditions, without complete relief of symptoms. They were completely relieved after repair of the hernia (Fig. 2).

The chief symptoms of esophageal hiatus hernia are pain, distress, gaseous eructation, vomiting, dyspnea, hemorrhage, weakness, anemia and palpitation of the heart. At the onset the symptoms are usually mild; they consist of epigastric distress that is projected through to the back and which comes on in the course of, or shortly after, a heavy meal. However, such attacks may be brought on by taking anything into an empty stomach, such as a cupful of coffee. The attacks are usually similar to one another in character but vary a great deal in intensity, depending on the amount of stomach that becomes incorporated in the hernia and the degree of interference with the diaphragm as well as the size of the hernial orifice and the occurrence of associated complications such as traumatic ulcer and incarceration of the stomach (Fig. 3).

In cases in which surgical treatment of the hernia is considered, one of the most important groups is that in which the symptoms simulate angina pectoris, for there are often no definite findings on which the diagnosis of coronary disease can be established. It is to be remembered that although a patient has a definite esophageal hiatus hernia that could adequately explain the symptoms, the patient can also have coronary sclerosis without proved signs and, if this condition is present, it constitutes a marked hazard to surgical intervention for the hernia.

From the standpoint of management hiatus hernia may be divided into three groups: In the first group the hernia is small, is recognized roentgenologically, often during the course of a general examination, and causes few or no clinical symptoms. No treatment is indicated in this group of cases. The second group includes those cases in which the symptoms are moderate and the herniae are of moderate size; in many of the cases in this group, conservative treatment, such as regulation of diet and reduction of weight, is sufficient to relieve the symptoms. The third group includes those cases in which there is no response to conservative measures; in these cases the herniae usually are large, and in many cases, in my experience, there are complications, such as incarceration of the stomach or gastric erosion. In this group of cases the only treatment that assures relief of symptoms is operative repair of the hernia.

In all cases in which a third or more of the stomach is involved in the hernia, surgical intervention should be considered, because the condition is progressive and usually the progressive enlargement becomes more rapid after the hernia has attained this size. Operation should be performed before severe incarceration, with consequent obstruction and traumatic lesions of the stomach, has occurred. The operative risk is increased by gastric reten-

tion and the technical difficulties are enhanced by fixation of the stomach to the diaphragm and to the hernial sac within the thorax. In all cases in which the colon is involved in the hernia, early operation is necessary because of the danger of occurrence of intestinal obstruction.

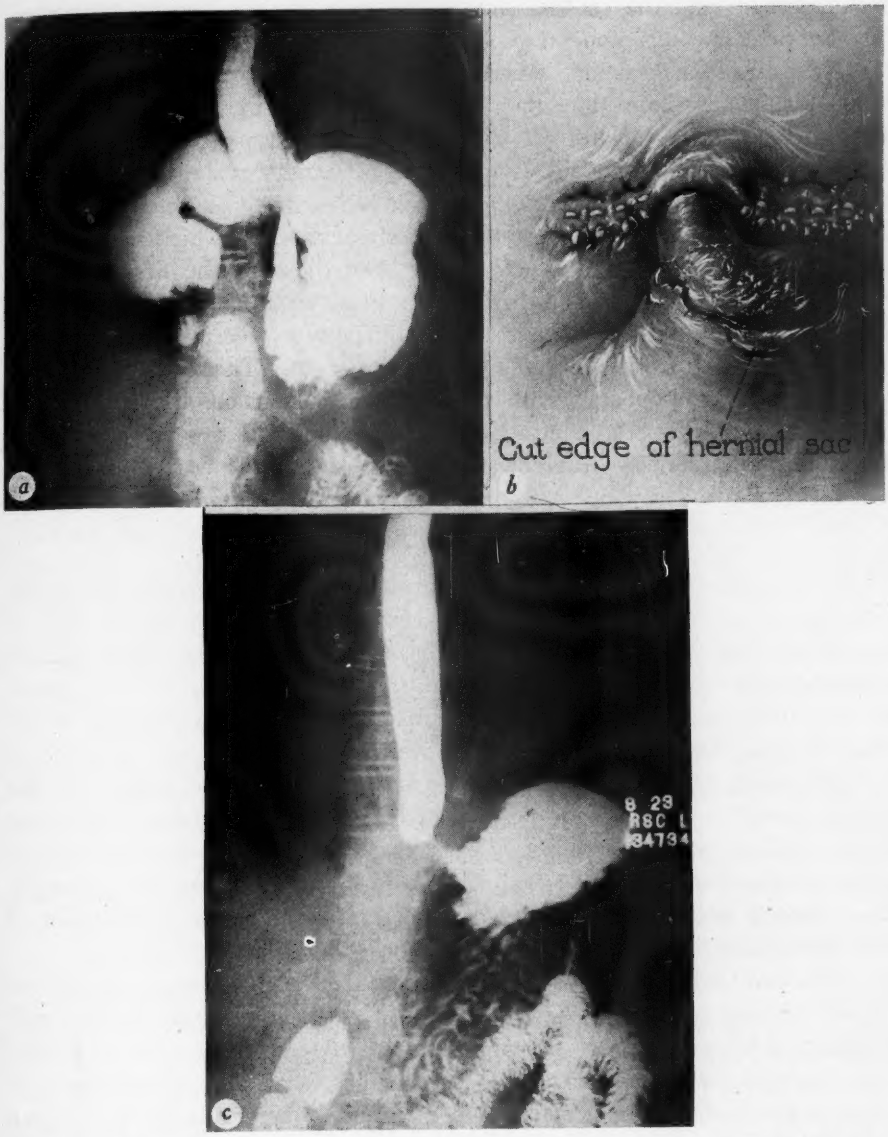

FIG. 4*a*.—Patient, age 55. Esophageal hiatus hernia with herniation of the entire stomach, which is inverted, and of a portion of the duodenum, with marked elevation of the esophagus and also herniation of the transverse colon through the esophageal hiatus.

b. Same patient. Repair of the markedly enlarged esophageal hiatus by overlapping the anterior margin over the posterior margin on both sides of the esophagus and at a higher level on the esophagus. Interrupted silk sutures and continuous sutures of fascia lata were used in the repair.

c. Same patient three weeks after repair of the hernia, showing the entire stomach in normal position below the diaphragm, which is slightly elevated as a result of interruption of the phrenic nerve. The esophagus is in normal position and extends to the diaphragm.

While all herniae through the esophageal hiatus are considered under the general term "esophageal hiatus hernia," there are three different types which are important from the standpoint of surgical technic. The first type consists of those cases in which the esophagus maintains its attachment to the diaphragm and the cardiac end of the stomach has herniated through the abnormal opening along the side of the esophagus. This is commonly called a para-esophageal hiatus hernia. It is, however, relatively infrequent and does not constitute more than 20 to 25 per cent of cases in which the patient comes to surgical treatment. The second type consists of those cases in which the esophagus is markedly retracted or shortened into the mediastinum but in which it is long enough to reach the diaphragm by traction. This type constitutes 75 to 80 per cent of cases in which treatment is surgical. The herniae are usually larger than those of the first type and the results are not as favorable from a surgical standpoint, for recurrences are more prone to develop because of the difficulty of reëstablishing fixation of the lower part of the esophagus to the diaphragm. The third type is that of the true short esophagus with partial thoracic stomach, which may also include cases of cicatricial contraction with fixation of the esophagus. These cases present an entirely different surgical problem from the true esophageal hiatus hernia.

SURGICAL TREATMENT

In most cases of esophageal hiatus hernia, I prefer to perform temporary interruption of the phrenic nerve by crushing the nerve preliminary to abdominal repair of the hernia because permanent paralysis of the diaphragm is rarely necessary in this type of hernia. Following temporary interruption of the phrenic nerve the function of the diaphragm is usually reëstablished in from three to six months. In cases in which reëstablishment of function of the diaphragm is not desired because of the danger of recurrence of the hernia, paralysis can be made permanent by cutting or evulsing the phrenic nerve. As a procedure preliminary to radical surgical treatment, interruption of the phrenic nerve is often of value in treatment of incarcerated and strangulated herniae because it prevents spasm of muscle and causes relaxation of the hernial ring.

Permanent interruption of the phrenic nerve may be a necessary procedure in the surgical treatment of partial thoracic stomach resulting from a congenitally short esophagus. However, I wish to emphasize that permanent interruption of the phrenic nerve is rarely necessary and should never be done in cases of hiatus hernia until it is definitely ascertained that it is not advisable to reëstablish the function of the diaphragm. It should be emphasized also that this procedure cannot replace the operative repair of the hernia. It is important to bear in mind that the atrophy of the diaphragmatic muscle which follows permanent interruption of the phrenic nerve may make it impossible to obtain a satisfactory result in the event of further radical repair of the hernia.

RADICAL SURGICAL REPAIR

I prefer the abdominal approach in all cases of esophageal hiatus hernia because the herniated viscera are contained in a sac in the posterior mediastinum and do not enter the true pleural cavity. An oblique incision is made in the left rectus muscle and peritoneum, extending to the ensiform cartilage. The technical difficulties of adequate exposure of the esophageal hiatus are often considerable because of fixation of the left lobe of the liver to the leaf of the diaphragm. The exposure of the hiatus is greatly facilitated by cutting the suspensory ligament and retracting the left lobe of the liver to the right. This can be accomplished, when the left lobe is small, by folding it on itself, and when it is large, by retracting it forward into the incision. The spleen is often very adherent to the posterior part of the diaphragm and hernial opening but usually can be separated from these structures by blunt dissection. It is retracted posteriorly by a specially constructed retractor. In some instances the spleen may be almost drawn into the hiatus and may be so traumatized by separating it from its peritoneal attachments that its removal is advisable.

Herniae through the esophageal hiatus are true herniae and have an hernial sac consisting of abdominal peritoneum which is continuous with the serosa of the stomach. The attachment of the sac to the stomach must be separated and the sac must be either completely removed or permitted to retract into the posterior portion of the mediastinum. I believe that this is one of the most important technical considerations in the surgical treatment of this type of hernia.

After the sac has been removed, the enlarged defective esophageal hiatus is repaired by overlapping the margins of the opening. In many instances it is necessary to elevate the repaired hiatus to a higher position on the esophagus. The latter is a very important procedure in those cases in which there is any shortening of the esophagus or marked elevation of the esophagus into the thoracic cavity (Fig. 4). Repair is usually made to the left of the esophagus but in some cases it is necessary to repair the opening partially both to the right and to the left of the esophagus. In some instances the defect of the esophageal hiatus is posterior, extending to the spinal column. This type requires the overlapping of the margins posterior to the esophagus. In such cases, the condition is often thought to be a herniation through the aortic opening but extending over the aorta there usually is an imperfectly developed, fibrous band which is the margin of the defective esophageal hiatus. The closure is usually made with living sutures of fascia lata, which are removed from the thigh. The overlapped margins of the hernial opening are first stabilized with interrupted linen sutures. The fascia lata is then woven into the tissues by continuous suture and fixed in the tissues with interrupted linen sutures.

In many instances in which the stomach is incarcerated or obstructed, it is impossible to pass a stomach tube into the obstructed loculus of the stomach before operation. In these cases it is advisable to pass a stomach tube

soon after the abdomen is opened, directing the tube into the obstructed portion of the stomach in order to empty the gastric contents before any attempt is made to reduce the herniated viscera, because of the danger of regurgitation and aspiration of gastric contents into the lung. Before closure of the defective esophageal hiatus is completed around the lower part of the esophagus, it is important that a stomach tube of large caliber be passed through the esophagus into the stomach, to aid in the reconstruction of the normal esophageal opening and to prevent constriction of the esophagus by a tight closure. The loose areolar tissue or a small portion of the esophageal wall at the cardia is incorporated into the innermost margin of the closure by a suture of chromic catgut.

The abdomen always should be thoroughly explored for the presence of any other lesion, particularly of the stomach or gallbladder. In some cases it may be necessary to operate upon other associated lesions. However, I do not believe it advisable to carry out any additional surgical procedure at the time of repairing the hernia, unless it is imperative, but it is well to know whether the patient has gallstones or any other lesion in the upper part of the abdomen which might account for subsequent symptoms.

Inasmuch as the surgical treatment of this type of hernia is a repair of an abnormally large hiatus of the esophagus and not a complete closure of an abnormal opening, it is associated with a higher percentage of recurrences than any other type of diaphragmatic hernia, in fact, nine out of ten recurrences in the entire series of 404 cases were of this type of hernia. In four of the nine recurrences, symptoms were severe enough to require a second operation. There were seven deaths (2.2 per cent) in the 320 cases of esophageal hiatus diaphragmatic hernia.

Congenital Short Esophagus.—The surgical treatment of congenital short esophagus with partial thoracic stomach presents an entirely different technical problem from that of esophageal hiatus diaphragmatic hernia. As pointed out previously, the essential consideration in the surgical treatment of esophageal hiatus hernia is that of replacement of the herniated stomach into the abdomen, the removal or obliteration of the hernial sac and the repair and reconstruction of the esophageal hiatus accurately around the esophagus.

Congenital short esophagus with partial thoracic stomach is not a true hernia through the diaphragm, in that the stomach has never been in its normal position below the diaphragm because of shortening of the esophagus. The surgical problem in these cases is that of reconstructing the diaphragm over the elevated portion of the stomach; this can be accomplished if the shortening of the esophagus is not too great. By complete and permanent interruption of the phrenic nerve the diaphragm usually can be elevated from 2 to 5 cm. and then by complete separation of the attachment of the esophagus from the attachments around the esophageal hiatus, from 2 to 3 cm. of the esophagus can be drawn down into the abdomen. The elevation of the diaphragm and the pulling down of as much as is possible of the esophagus into the abdomen permit the esophageal hiatus to be closed around the lower

end of the esophagus, placing what was formerly the thoracic portion of the stomach below the diaphragm.

TRAUMATIC DIAPHRAGMATIC HERNIA

The causation of traumatic diaphragmatic hernia has been considered in an earlier section of this paper.

Traumatic diaphragmatic herniae usually do not present the difficult diagnostic problems which are associated with the esophageal hiatus type of hernia, for the occurrence of the injury leads one to suspect the possibility of a hernia being present. The symptoms associated with this type of hernia progress very rapidly, are severe, and are attributable to the mechanical interference with the function of the herniated viscera as well as to marked interference with function of the heart and lungs. This is due to the fact that there is no hernial sac and the abdominal viscera are in direct contact with the thoracic viscera. The condition in these cases may be more properly termed "evisceration of the abdominal organs into the pleural cavity" rather than a "true hernia." The most marked immediate symptoms are usually those of respiratory and circulatory embarrassment. These herniae are more frequent in adult life and the compensatory cardiac and respiratory reserve usually carries the patient over the acute symptoms if the other associated injuries have not been too great. Later, severe hemorrhage from the gastro-intestinal tract may occur as a result of incarceration or strangulation of the hollow viscera. If the patient survives the acute condition, the later symptoms depend on the viscera involved. The symptoms may consist of obstinate constipation, the occurrence of large quantities of gas in the colon and attacks of partial or complete intestinal or gastric obstruction. The sudden onset of symptoms in cases of traumatic hernia usually is related directly to the injury and there is rarely a question as to the clinical diagnosis. Surgical treatment is demanded because of the danger of cardiac and respiratory failure or because of intestinal strangulation (Fig. 5).

Those types of diaphragmatic hernia which result from inflammatory necrosis of the diaphragmatic muscle caused by subdiaphragmatic abscess or pressure from the drainage tubes used for drainage of empyematic cavities, are considered as traumatic herniae. The symptoms associated with these herniae are often somewhat obscure and in many instances they are unrecognized for a long period of time because the possibility of a hernia is not considered and the symptoms are often thought to be due to the primary illness. In some instances the hernia does not occur in these cases for many months after the patient recovers from the primary illness.

The surgical approach to these herniae may be through the thorax or through the abdomen. For all herniae through the right side of the diaphragm, I prefer the thoracic approach because the large right lobe of the liver interferes with the exposure of the right side of the diaphragm if the abdominal approach is used. In herniae of this side the right lobe of the liver is often incorporated in the hernia and its reduction is more safely accomplished

through the thoracic approach than through the abdominal approach because there is less danger of hemorrhage from injury to the liver.

In all traumatic herniae through the left side of the diaphragm, I prefer the abdominal approach through an oblique left rectus incision. The herniated viscera are usually very adherent to both the abdominal and the thoracic side of the diaphragm and to the structures within the thorax. The adhesions

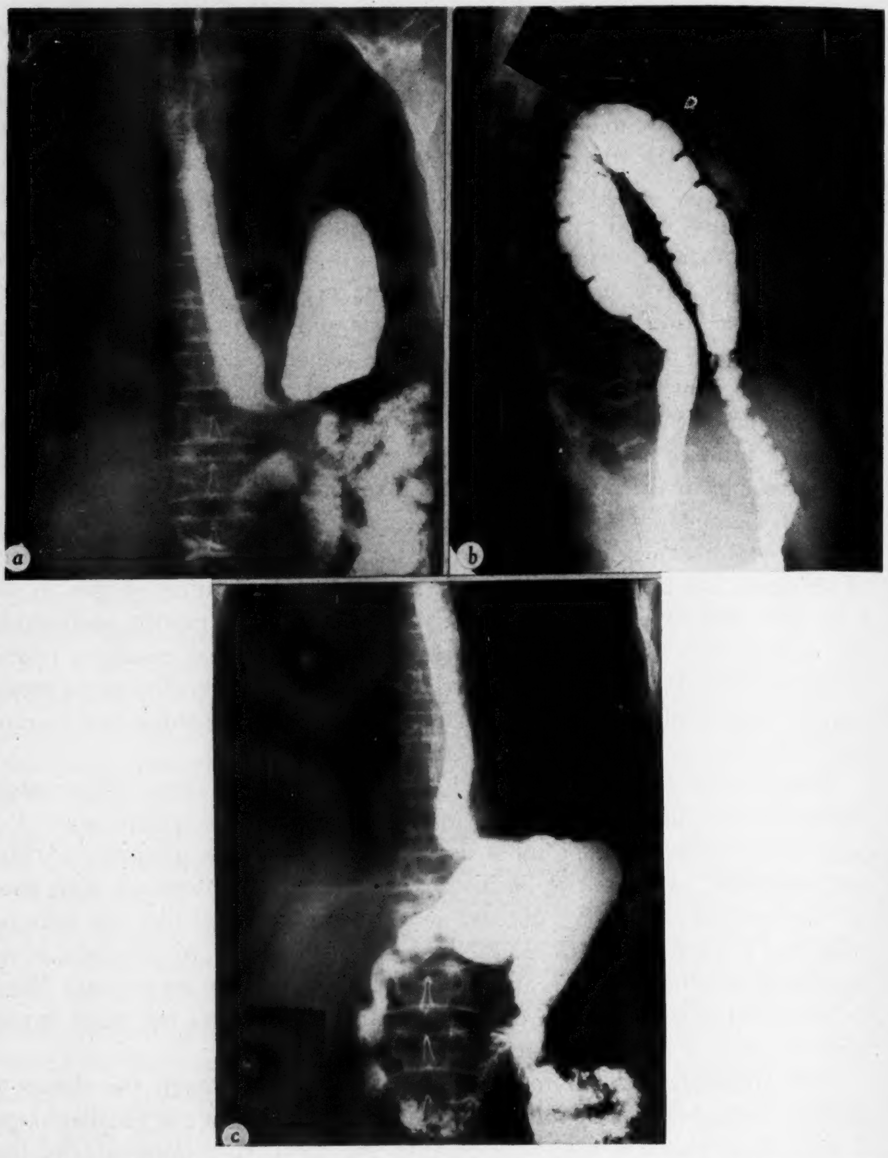

FIG. 5*a* and *b*.—Patient, age 73. Large left traumatic diaphragmatic hernia due to an automobile accident. Herniation of the entire stomach, transverse colon and spleen and of several feet of small bowel. The esophagus is in normal position.

c. Same patient three and one-half weeks after repair of laceration in the posterior portion of the left diaphragm which did not involve the esophageal hiatus. The entire stomach is in normal position below the diaphragm.

to the margins of the opening and to the under surface of the diaphragm are often very marked and should be separated first. The adhesions to the structures within the thoracic cavity are separated from below upward by approaching them through the hernial opening. By the abdominal approach this can be accomplished with little danger of injury to the abdominal or thoracic viscera, because the definite relationship of the herniated structures can be established.

In cases in which there has been considerable loss of structure or in which the muscle has been torn from its attachment to the thoracic wall, the defect in the diaphragm should be repaired by fascia lata stabilized with linen sutures. I believe this to be the most satisfactory type of closure in all these cases. In cases of traumatic hernia in which the laceration is confined to the dome of the diaphragmatic muscle, it usually is advisable to repair the opening by lapping the anterior margin over the posterior margin of the opening. When possible, it is advisable to overlap the margins of the opening from 2 to 3 cm. In those cases in which the laceration splits the muscle of the esophageal ring, great care should be taken in repairing the esophageal hiatus. In those cases in which the laceration extends to the margin of the thorax and in which the attachments of the diaphragm are torn from the thoracic wall, the repair is made not only by overlapping the laceration of the leaf of the diaphragm but by resuturing the diaphragmatic muscle to the thoracic wall. This can be accomplished by suturing the diaphragmatic muscle to the intercostal muscles between the ribs. When possible, the diaphragmatic muscle should span two interspaces, being fixed to the intercostal muscles with fascia lata and stabilized with interrupted linen sutures.

In a few instances the relaxation of the diaphragmatic muscle caused by interruption of the phrenic nerve will not be sufficient for repair of the defect. In these cases the diameter of the thorax must be narrowed by resecting the lower ribs by thoracoplasty. It is usually not necessary to resect more than a few inches of the eighth, ninth and tenth ribs at the angles.

Before the abdomen is closed, the herniated viscera should be thoroughly explored, to be certain that there has been no injury to a viscus and that there are no bands of adhesions which will interfere with the function of the abdominal viscera. In cases in which there has been considerable obstruction of the large bowel, it may be necessary to perform appendicostomy or colostomy at the time of operation.

In the series of 55 cases of traumatic diaphragmatic hernia there were no recurrences, and four deaths.

CONGENITAL DIAPHRAGMATIC HERNIAE DUE TO MALFORMATION AND STRUCTURAL DEFICIENCIES

Congenital diaphragmatic herniae may occur in either the right or the left side of the diaphragm but are much more common through the left side than through the right. The more common herniae of this type are those

through the pleuroperitoneal hiatus, those due to the lack of formation of the posterior portion of the diaphragm and those through the foramen of Morgagni (Larrey's space), anteriorly, more accurately termed subcostosternal herniae.

In the first two types there is rarely, if ever, an hernial sac and the abdominal viscera are in direct contact with the thoracic viscera. In the third type (subcostosternal) there is always an hernial sac, which consists of peritoneum and parietal pleura.

The symptoms of congenital types of diaphragmatic hernia due to structural deficiency in the formation of the diaphragm usually involve multiple abdominal viscera and are often similar to those noted in association with the traumatic types of hernia, as there is rarely a confining sac and the herniated abdominal viscera are in direct contact with the thoracic viscera. The symptoms in these cases are often more severe than those noted in cases of traumatic hernia. Because of the occurrence of the hernia at birth, the respiratory and cardiac symptoms are usually the most severe owing to the marked unilateral alteration in intrathoracic pressure and the occurrence of this derangement of intrathoracic pressure at a time at which the compensatory respiratory and cardiac reserve has not been developed to a sufficient degree to maintain function of these organs. Many infants born with these congenital defects die in the first few hours or days of life. However, if the respiratory and cardiac mechanisms are able to compensate for the presence of these abdominal viscera in the thorax, these patients may live on to childhood or even to adult life without any great amount of disability or symptoms, provided that intestinal or gastric obstruction does not develop. There is less likelihood of obstruction developing in these cases than in the cases of traumatic hernia because there are usually fewer adhesions between the abdominal viscera and the thoracic viscera in the former than in the latter. When the stomach is involved in these herniae, it usually becomes markedly dilated and these patients often have symptoms of partial gastric obstruction. Intestinal obstruction may occur owing to bands of adhesions between the omentum and loops of bowel or owing to inflammatory conditions of the bowel. Inasmuch as there is usually a nonrotation of the right portion of the colon and the cecum, and the appendix is in the left thoracic cavity, appendicitis may develop and produce a very serious hazard to life.

In the surgical treatment of these herniae the approach in the first two types may be either thoracic or abdominal but I prefer the abdominal approach through an oblique left rectus incision. In the third type (substernal) the approach should always be through the abdomen and usually through an oblique right rectus incision or a transverse incision in the epigastrium. I prefer the oblique right rectus incision.

Pleuroperitoneal Hiatus Herniae.—These herniae occur in the posterolateral portion of the diaphragm and are due to failure of fusion of the pleuroperitoneal membrane and the septum transversum. The defect is usually triangular with the apex toward the median portion of the diaphragm.

The defect usually extends to the thoracic wall but occasionally there is an imperfectly developed band of muscle tissue extending along the thoracic wall. These herniae do not have an hernial sac and there is a direct communication between the abdominal and the thoracic cavity.

The most common abdominal viscera involved in this type of hernia are the colon and the small bowel. There may or may not be herniation of the

FIG. 6*a* and *b*.—Patient, age four months. Pleuroperitoneal hiatus hernia with herniation of many loops of large and small bowel in the left thoracic cavity. Marked displacement of the mediastinum and heart to the right. The stomach is dilated and below the diaphragm.

c. Same patient on reëxamination one year after operation. The stomach and the intestines are entirely below the diaphragm, which is of normal contour and position. The heart and the mediastinum are in normal position. Both pulmonary fields are normal.

spleen and stomach. There is often a failure of rotation of the colon and the entire right side of the colon (appendix and cecum), the terminal part of the ileum and all of the small intestines to the jejunum are involved in the hernia.

This type of hernia is said to be the most common of the congenital types of hernia due to structural deficiencies. These herniae are present at birth. Many of the infants suffering from them die in the first few hours or days of life because of respiratory and cardiac embarrassment and before surgical intervention can be instituted. In treating those infants who are able to survive in spite of the altered intrathoracic pressure and thoracic visceral relationship, surgical intervention should be instituted as soon as possible because of the danger of intestinal obstruction. If they are able to maintain nourishment, it is well to delay operation for two to three months in order to permit some development of their accessory respiratory mechanism. If operation is delayed for a long period, the abdominal viscera will have lost their right of residence in the abdomen in that the abdominal cavity will not have developed sufficiently to contain them and there will be marked increase in the intra-abdominal pressure when the viscera are replaced into the abdomen (Fig. 6).

In repair of the smaller herniae of this type the opening can be closed without utilizing interruption of the phrenic nerve. On the other hand, in repair of the larger herniae interruption of the phrenic nerve is a necessary procedure. The opening is completely closed by overlapping the margins from 2 to 3 cm. If the patient is an infant, this closure is made with interrupted silk sutures. Before the opening is completely closed, the air is aspirated from the pleural cavity by inserting a catheter connected to a suction apparatus. At the time of withdrawal of the catheter the last suture is tied, completely closing the communication between the thorax and the abdomen.

One of the chief dangers associated with the repair of these herniae is marked alteration of intrathoracic or intra-abdominal pressure. It is very important in these cases that the respiratory function be maintained by positive pressure during the operation and that at the completion of the operation negative pressure be obtained and secured in the thoracic cavity. A roentgenogram should be taken at the completion of the operation to see that there is no shift of the mediastinum due to the pneumothorax. I do not permit the patient to leave the operating table until I have seen the roentgenogram. If there is any shift of the mediastinum, more air is withdrawn to maintain the mediastinum in the midline.

In the series of nine cases of hernia through the pleuroperitoneal hiatus there were no recurrences and three deaths.

Congenital Absence of the Posterior Portion of the Diaphragm.—This type of hernia is due to failure of the formation of that portion of the diaphragm which is derived from the pleuroperitoneal membrane. The defect is in the posterolateral portion of the diaphragm and usually extends from the eighth rib posteriorly and medially to the esophageal hiatus. These

herniae usually do not have a sac but there may be an imperfectly developed enveloping membrane of peritoneum and omentum which simulates a sac. These herniae may be considered an enlargement of the foregoing pleuroperitoneal type in that the essential difference is a much more extensive congenital defect in the formation of the diaphragm. There are more abdominal viscera involved in the hernia in that these herniae always contain the stomach and spleen as well as the large and small bowel. Occasionally the left kidney is elevated above its normal level into the pleural cavity (Fig. 7).

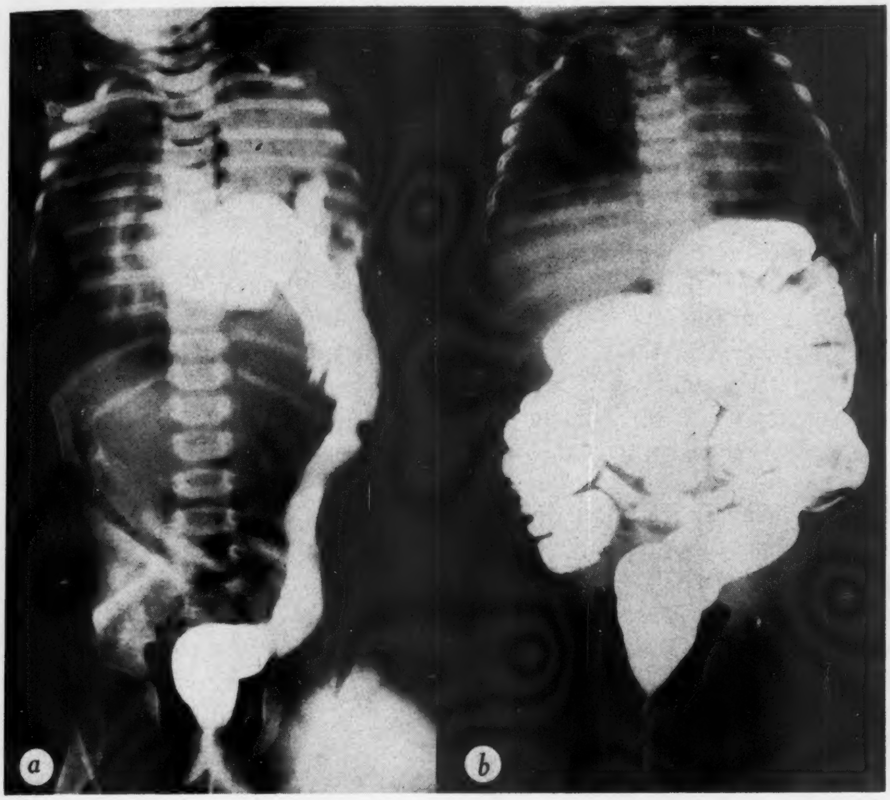

FIG. 7a.—Patient, age six months. Congenital absence of a portion of the diaphragm. Herniation of the entire right portion of the colon and cecum (small bowel and stomach), into the left thoracic cavity. Complete collapse of the left lung. Marked displacement of the heart and mediastinum to the right.

b. Same patient one month after reconstruction of the defective diaphragm (with fascia). Entire colon (and other herniated viscera) below the diaphragm. The left diaphragm, the heart and the mediastinum are in normal position. Pulmonary fields normal.

Not only do the surgical problems associated with these herniae involve all of the problems of the pleuroperitoneal herniae as far as altered intra-abdominal and intrathoracic pressure is concerned but in addition there is the problem of closing this large gap with the diaphragmatic muscle that is present and of reconstructing the attachment of the diaphragmatic muscle to the thoracic wall. In some instances the posterior perirenal fascia may be utilized in obtaining this closure and fixation to the thoracic wall. If the

gap is not too great, this can be accomplished by complete, permanent interruption of the phrenic nerve. If the defect is too large to permit the relaxed diaphragm to span this gap, it is necessary to shorten the diameter of the diaphragm by extrapleural rib resection.

In 12 cases herniae were due to congenital absence of a portion of the diaphragm. There were one recurrence, and two deaths.

Subcostosternal Hernia through the Foramen of Morgagni (Larrey's space).—Herniation of abdominal viscera through regions of deficiency of muscle in the anterior portion of the diaphragm close to the sternum has received various names, such as diaphragmatic hernia through the foramen of Morgagni, or through Larrey's fissure or space, and also substernal, retrosternal, parasternal or anterior diaphragmatic hernia. Inasmuch as these herniae usually occur to either side of the anterior midline of the diaphragm, if an anatomic term is to be used, it would be preferable to designate them as subcostosternal diaphragmatic herniae.

There is some difference of opinion as to whether these herniae should be classified as congenital or acquired herniae. It is impossible to explain their occurrence on a basis of faulty fusion or improper disposition of the embryonic mesodermic elements which go to form the diaphragm, as this anterior portion of the diaphragm is derived from the septum transversum only. But the consistency of the location of the hernial opening, the fairly constant relation of the neck of the hernial sac to the round and falciform ligaments of the liver and the frequency with which the hernial sac protrudes into the right side of the thoracic cavity at the same point of entrance at the cardiophrenic angle, as well as the often associated nonrotation of the right portion of the colon, all strongly suggest a fundamental embryologic basis for these herniae. These herniae are essentially direct herniae through a congenital defect in the structure of the diaphragm or a faulty attachment of the diaphragm to the sternum and costal cartilages. The constant presence of a peritoneal sac shows that the peritoneum had closed off the abdominal cavity from the pleural cavity before the actual herniation of the abdominal viscera occurred.

Subcostosternal diaphragmatic hernia is one of the two types of diaphragmatic hernia, in my experience, which have an hernial sac. The other type of diaphragmatic hernia which has an hernial sac is that through the esophageal hiatus. It is interesting that subcostosternal hernia is probably the rarest type of diaphragmatic hernia and esophageal hiatus diaphragmatic hernia is the most common; both are essentially congenital in origin but are rarely present at birth, and occur in most instances in later life because of increased abdominal pressure on a congenitally defective diaphragm.

The abdominal viscera usually involved in the hernia are the colon, omentum, ileocecal coil and rarely the stomach (Fig. 8).

The subjective symptoms associated with these herniae are often indefinite and depend on the type and amount of abdominal viscera involved in the hernia. They are usually due to impairment of respiration and intestinal

obstruction. Among them are dyspnea, cough and attacks of partial intestinal obstruction and thoracic and abdominal pain.

In the cases in which hollow viscera are involved in the hernia, symptoms occur which suggest the possibility of a hernia or at least the necessity of a roentgenologic examination of the intestinal tract which will determine whether a hernia is present. The cases in which omentum only is involved in the hernia present a much more difficult clinical problem in arriving at a

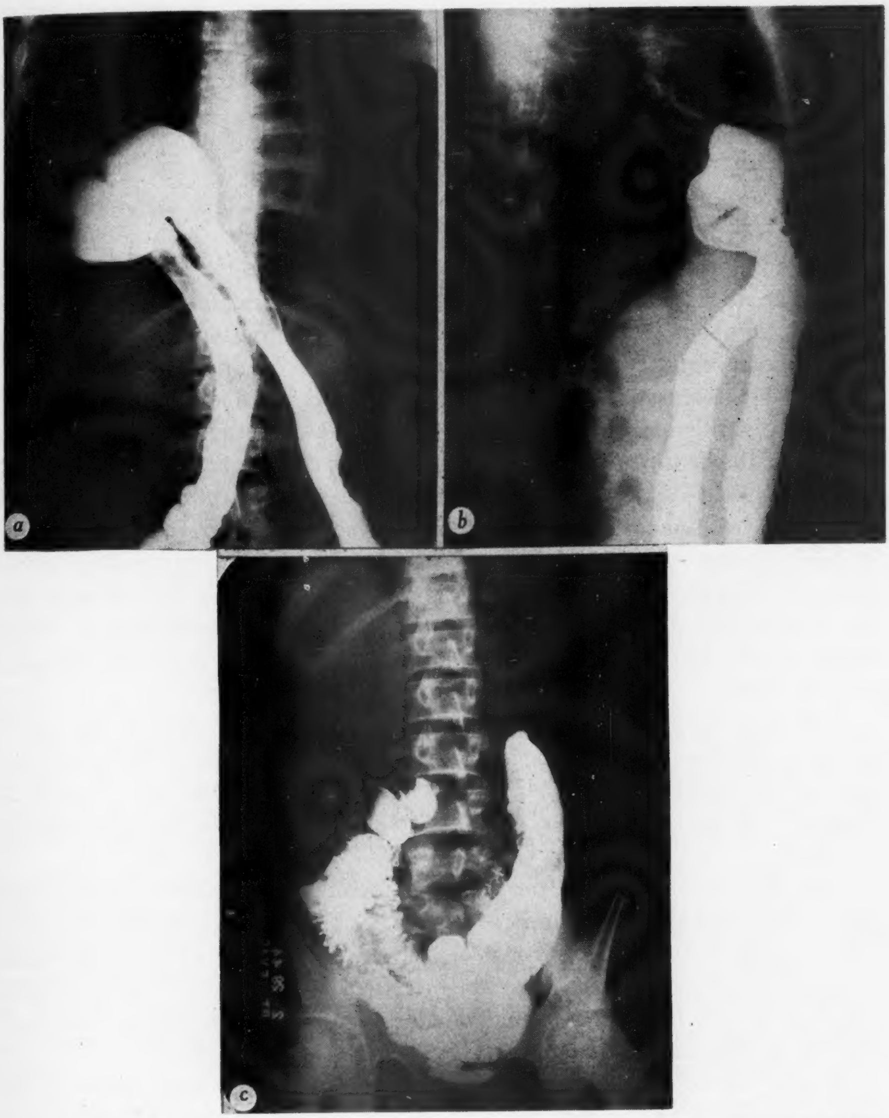

FIG. 8*a* and *b*.—Patient, age 35. Subcostosternal (foramen of Morgagni) diaphragmatic hernia. Herniation of transverse colon with marked displacement of splenic and hepatic flexures into the right anterior thoracic cavity at the cardiophrenic angle.

c. Same patient one month after repair of hernial opening in the anterior diaphragm. The entire colon is below the diaphragm.

definite diagnosis. The subjective symptoms in the latter group are entirely thoracic as a result of mechanical interference with respiration and expansion of the lungs. These symptoms suggest a primary pulmonary lesion and direct the clinical investigation to roentgenologic study of the thorax. The roentgenologic findings of an increased density in the pulmonary field justify the clinical diagnosis of a primary intrathoracic lesion which may be thought to be an intrathoracic tumor. This erroneous clinical diagnosis is particularly likely to occur if there are no subjective symptoms even to suggest that an abdominal condition may be present and, even though the gastro-intestinal tract is examined roentgenologically, no lesion is demonstrated as no abdominal hollow viscera are involved in the hernia.

One of the most important clinical considerations of this type of hernia is the possibility of regarding the patient's condition as due to an intrathoracic tumor in the cases in which the omentum is the only abdominal structure involved in the hernia.

The treatment of these herniae is surgical closure of the abnormal opening in the diaphragm after replacement of the abdominal viscera into the abdomen. I prefer an abdominal approach through the upper part of the right rectus muscle because the opening in the diaphragm is very accessible and the abdominal contents of the hernia are more safely and easily reduced from the abdominal than from the thoracic side of the diaphragm as the true relationship of the herniated viscera to the hernial sac can be accurately determined.

The method of closure of the neck of the sac and of the defect in the structure of the muscle of the diaphragm depends on the size and character of the opening. Small linear openings may be closed by overlapping the margins. Larger transverse openings extending beneath the sternum are best closed by suturing the anterior margin of the diaphragmatic muscle defect to the posterior sheath of the rectus muscle and to the anterior thoracic wall.

The most satisfactory material for closure of the opening is living suture of fascia lata removed from the thigh and stabilized in the tissues with silk. The round ligament of the liver can be incorporated in this closure to strengthen it as well as to reëstablish its position on the anterior abdominal wall.

The closure of the large openings is facilitated by paralyzing the right side of the diaphragm by temporary interruption of the right phrenic nerve. This procedure, however, is not necessary in the closure of small openings. Preparation can be made to interrupt the phrenic nerve in the supraclavicular region after exploring the opening and determining whether or not interruption is necessary.

In this series of 404 cases of diaphragmatic hernia, eight were of the subcostosternal type. There were no deaths or recurrences following their operative treatment.

In Table II the surgical procedures and operative results in the entire series of 404 cases are given.

CONGENITAL DIAPHRAGMATIC HERNIA*

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In reviewing the literature of the past ten years on congenital diaphragmatic hernia, one is as impressed by the number of single cases reported by various authors as well as by the series of cases, such as those of Harrington,[7] Ladd and Gross,[12] and Hartzell,[8] and Truesdel,[15] Many of these cases have been operated upon successfully at an early age. It is obvious from these reports that the diagnosis is made much earlier and that the operative treatment has made great progress in recent years. In 1925, Hedbloom reported that 75 per cent of congenital herniae died before they were one month old. In 1938, I reported ten cases from the Babies Hospital, six of whom had been operated upon. The purpose of this paper is to review the surgically-treated cases of the first series, add to their follow-up, and to report 11 additional cases operated upon since 1938.

It is still difficult to estimate the incidence of congenital diaphragmatic hernia because of the dissimilar reports, but the percentage of cases found in a given number of roentgenologic examinations seems to be steadily increasing. Bradley, from the Mayo Clinic, reported the occurrence as 1 in 18,000 cases. In 1920, MacMillan found only three cases in 15,000 roentgenologic examinations, and in 1924, Pancoast and Boles reported 16 cases in 9,000 gastro-intestinal series. In 1938, Dickson reported 206 cases during a 14-year period from the Toronto General Hospital. I am quite sure that the condition occurs much more frequently than has been suspected.

The most common congenital defects in the diaphragm are, in order of their occurrence: (1) Esophageal hiatus. (2) Foramen of Bochdalek. (3) Foramen of Morgagni. (4) Defects in the dome. Herniae through the vena caval or aortic openings in the diaphragm have never been reported.

EMBRYOLOGY

The complexity of the embryonic development of the diaphragm predisposes it to congenital defects, most of which result from the failure of fusion of the component fibers at various points. The defects in the dome of the diaphragm are more difficult to explain from an embryologic standpoint other than to say that they are due to a failure of fusion at this point. The embryonic diaphragm consists of two parts: a ventral part which is the cephalic portion of the septum transversum, developing in the cervical region; and a dorsal part which is the pleuroperitoneal membrane, developing from the lateral body walls and destined to become the closing membrane between the pleural and the peritoneal cavities. The muscular portion of the diaphragm develops while it is in the cervical region from the third and fourth cervical myotomes on each side. During development, the diaphragm migrates from the region of the third cervical vertebra to its final location opposite the

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twelfth thoracic vertebra, and, during this migration, its plane of direction changes many times. It is thought that the original communication between the pleural and peritoneal cavities closes about the third month of intra-uterine life. If this communication remains open, there is formed the pleuroperitoneal hiatus known as the foramen of Bochdalek. Since the liver lies over the right foramen, herniae occur more often on the left than on the right side. Failure of fusion of the costal and sternal fibers at either side of the sternum results in the formation of the foramina of Morgagni. The defects in the dome are more common on the left side. Hernia through the esophageal hiatus has been attributed either to the failure of development of the diaphragm at that point, or to the failure of migration of the stomach because of a short esophagus. These herniae usually have a peritoneal sac. While herniae through the esophageal hiatus and through the foramen of Morgagni usually have a peritoneal sac, herniae through the foramen of Bochdalek usually do not, and defects in the dome of the diaphragm may or may not include one.

SYMPTOMOLOGY

The symptoms of diaphragmatic hernia may be either circulatory, respiratory, gastro-intestinal, or a combination of all three. They are due to mechanical interference with the function of the herniated structures or to interference with respiratory or circulatory organs upon which the herniated structures encroach. In most of the cases of hernia through the foramen of Bochdalek, the usual finding is that the chest is full of intestines, both large and small, often accompanied by part of the stomach and, quite frequently by the spleen and kidney. The symptoms in such cases are quite sure to be cyanosis, dyspnea, nausea, vomiting and signs of incomplete intestinal obstruction. On the other hand, it is surprising to find how few symptoms some of these patients have even though many of the abdominal structures are in the chest. One patient in this series had a little difficulty breathing during the hot weather; another had been perfectly well, and the diaphragmatic hernia was discovered during a routine physical examination. One patient had failed to gain weight as rapidly as normal; another had a cough, diarrhea and tarry stools for several weeks. As the symptoms vary to such a marked degree, the diagnosis of diaphragmatic hernia should always be considered when patients exhibit perplexing upper abdominal, respiratory or cardiac symptoms.

DIAGNOSIS

The diagnosis of diaphragmatic hernia may be made easily from a roentgenogram of the chest, which will show the presence of gas bubbles, collapsed lung and displacement of the heart away from the affected side. The roentgenologic examination has probably been the greatest factor in the discovery of many diaphragmatic herniae. If opaque media are used, it is not only possible to identify the herniated structures, but a lateral view of a barium enema will demonstrate whether the defect in the diaphragm is in front of or behind the liver. Without roentgenograms, the diagnosis will

often be missed since the physical signs may be confusing, and vary with the number of abdominal structures in the chest. Many cases of diaphragmatic hernia have been diagnosed as dextracardia, congenital heart, pneumonia, empyema and tuberculosis. To prove how confusing the physical signs may be, without roentgenograms of the chest, I would like to cite the difficulties in the diagnosis of one of the cases reported here. When I saw the patient first, she was two years old and had "lung trouble since birth." She had just completed a four-month period of bed rest because the parents were told that she had "adult type of pulmonary tuberculosis." Many other diagnoses had been made, such as empyema, unresolved pneumonia, *etc.*, and her chest had been explored with a needle 15 times. She had been seen by a number of doctors but had never had a roentgenogram taken of her chest. Her last physician heard signs in the chest which he interpreted as being due to the presence of intestinal coils and had a roentgenogram taken which confirmed his diagnosis.

TIME FOR OPERATIVE INTERVENTION

Operative repair is the only relief for this condition. I agree with Dr. Ladd, who has stated that these cases should be operated upon as quickly as possible after the diagnosis is made. We have followed his example in many of our cases, operating as soon as we felt reasonably sure the baby could tolerate it. Recently, we have taken two babies to the operating room in Davidson beds, which provide oxygen, as neither of them could be kept out of oxygen for more than a few moments. They were anesthetized, an intratracheal catheter introduced, and the operation proceeded without the least difficulty. When the chest contains either small or large intestine, the operation should be performed immediately as such patients are apt to develop intestinal obstruction which is a most unfortunate complication, as we found in one of our early cases (Case 3). While attempting to improve this baby's condition which was too poor to stand operation, he developed a high intestinal obstruction. He had to be operated upon at once since his obstruction was high in the jejunum, and, at operation, the distended loops were the last ones to be removed from the chest. He died 12 hours after his operation in spite of high concentration of oxygen, transfusions, *etc.* Another reason for not delaying operation is that the abdomen will not develop while the structures are allowed to remain in the chest, and when the structures are returned to the abdomen, it will be too small to receive them. We had this unfortunate experience in one of our cases who had had symptoms of diaphragmatic hernia since birth but was not operated upon until he was nine years of age.

OPERATIVE PROCEDURE

There is a difference of opinion as to whether the operative approach should be through the chest or the abdomen. Many cases reported in the literature have had both abdominal and thoracic incisions and often a combined thoraco-abdominal incision has been used. I prefer an abdominal approach through a subcostal incision, and have used it in all cases. In one

recurrent case, it was necessary for us to make a thoracic incision in addition to the abdominal incision. It has always seemed to me to be easier to reduce the structures by gentle traction from below than by forcing them down from above. The abdominal approach also lessens the possibility of postoperative respiratory complications and gives the operator an opportunity to replace the abdominal structures in about their normal positions. As a rule, there are no adhesions present in the chest unless the case is one of long standing, and the structures usually are easily reduced if the operator places a retractor in the diaphragmatic defect and equalizes the pressure in the chest and abdomen before he tries to reduce the structures. If he does not do this, he will find that the intestines will be sucked back in the chest about as fast as one can reduce them. Mattress sutures of silk should be used for repair of the defect overlapping the edges if possible.

In three cases of this series, while the defect was in the posterior part of the diaphragm, it differed considerably from hernia through the foramen of Bochdalek. There was a large opening, no attachment of the diaphragm to the posterior chest—probably a deficiency of the diaphragm. In these cases, I used a row of silk sutures with two needles on each, passing them through the diaphragm to the outside of the chest wall, one on either side of the adjacent rib and tied outside over a piece of gauze. Due to the deficiency of reparative material in these cases, the above row of sutures in closing the defect attached the diaphragm at a higher level in the chest than normal. A second row of continuous or interrupted silk then attached the edge of the diaphragm to the pleura and intercostal muscle. In any type of closure, a catheter is placed in the chest and the air removed before the last suture is tied. It is also desirable to have the phrenic nerve where it can be easily crushed if necessary. It has been our custom to expose the phrenic nerve in the neck a day or two before the repair, placing a traction suture about it so that it can be drawn out and crushed, if desired, during the repair of the diaphragm. We have done this in most of our cases except in the very young babies who were in bad condition. This exposure of the phrenic nerve is unnecessary, of course, if the thoracic approach is used. Intratracheal or positive-pressure anesthesia are desirable.

The difficulties of operation depend upon the location and size of the defect and upon the presence of adhesions. The mortality depends to a large extent upon the age of the patient.

POSTOPERATIVE CARE

All patients should be placed in an oxygen tent immediately after operation, and should be kept there for several days. The oxygen relieves abdominal distention, decreases both the respiratory efforts of the baby and the possibility of postoperative respiratory infection because of the easy regulation of the tent's temperature. Fluid requirements should be met by parenteral methods. In many cases, we have used a continuous intravenous drip to supply glucose and saline. Pleural effusion may occur and should be watched for, the chest being aspirated if necessary.

CASE REPORTS

GROUP I—HERNIA THROUGH THE FORAMEN OF BOCHDALEK

Case 1.—P. N., male, 4.5 months old, was admitted to the Babies Hospital, October 11, 1930, having had difficulty in breathing during the hot weather. He had had a convulsion 24 hours before admission, but was well otherwise. Roentgenologic examination showed small intestine in his left chest.

Operation.—The abdomen was opened through a left subcostal incision revealing a hernia through the left foramen of Bochdalek. His chest contained all of the small intestine beyond the duodenojejunal juncture, all the colon to a point beyond the splenic flexure, and the spleen. The splenic flexure was adherent to the parietal pleura, and was brought down after mobilization. All structures were reduced, and the diaphragmatic opening was closed with mattress sutures of black silk, the edges of the hiatus being overlapped. The patient was kept in an oxygen tent and had a continuous intravenous drip of saline for several days postoperative. His convalescence was uneventful.

Follow-up: Six and one-half years postoperative the child was normal in every way.

Case 2.—S. D., female, six months old, was admitted to the Babies Hospital, November 13, 1931, having vomited during the preceding five days. Her birth was full-term by cesarean section, and she had gained steadily, with no illness previous to the present one. She was well-developed and well-nourished. A gastro-intestinal series showed small and large intestine in the right chest.

Operation.—November 20, 1931: The abdomen was opened through a right subcostal incision. On retracting the liver from the diaphragm, a patent foramen of Bochdalek was revealed behind it. The chest contained the third and fourth parts of the duodenum, all the small intestine, and all the large intestine as far as the midtransverse colon. The structures were reduced, and the opening closed with mattress sutures of black silk. Convalescence was uneventful.

Follow-up: Roentgenologic examination six years postoperative showed the lung expanded and all abdominal structures below the diaphragm. The child has been very well.

Case 3.—M. B., male, age five weeks, was admitted to the Babies Hospital, January 6, 1936, with a history of vomiting and cyanotic episodes for two days. "Was developing well until two days ago when he collapsed, turned blue and had rapid respirations." He had had a similar spell 24 hours before admission. He had been delivered by cesarean section. Roentgenologic examination showed the right chest filled with small intestine. While under observation, the patient developed a high intestinal obstruction and was immediately operated upon.

After isolation of the phrenic nerve in the neck, a right subcostal incision was made. A large, posterolateral defect was found in the diaphragm through which practically all of the small intestine had herniated into the chest. A definite obstruction, due to a kink, was found in the upper jejunum. The opening in the diaphragm was closed with mattress sutures of black silk. The patient was placed in an oxygen tent immediately after operation, but he died 12 hours later as the result of shock.

There was no choice but to operate upon this case. A palliative operation could not be performed because of the high intestinal obstruction. This is a serious complication, and one which may happen in any case where there is either small intestine or colon in the chest. This patient also had an umbilical hernia, bilateral cryptorchidism, penile hypospadias, hypertelorism, and a pilonidal sinus.

Case 4.—E. C., male, age ten months, was first admitted to the Babies Hospital, in 1924, with a history of vomiting and convulsions. Roentgenologic examination showed

the whole left chest filled with small intestine. He was discharged but was readmitted several times subsequently, with signs of an intestinal obstruction. Each time he was taken home against advice, when his obstructive symptoms had disappeared. He was finally admitted, when eight years old, with obstructive symptoms, at which time his parents consented to an operation.

FIG. 1.—Autopsy specimen of hernia through the left foramen of Bochdalek. Small and large intestine in the left chest. Heart displaced to the right, both lungs compressed by the herniated structures.

Operation.—April 25, 1932: The abdomen was opened through a left subcostal incision, which revealed a patent, left foramen of Bochdalek as well as a defect in the left dome. The left chest contained all of the small intestine from the duodenojejunal juncture on, all of the colon to a point beyond the splenic flexure, and the spleen. The structures were removed from the chest after a very tedious dissection. Both openings in the diaphragm were closed with mattress sutures of silk. The structures could not be returned to the abdomen because they had never been there before, and the cavity was not large enough to contain them. The child's condition was desperate in spite of a transfusion and an infusion on the operating table. His condition was so precarious that the defects in the diaphragm were reopened and the viscera replaced into the chest. The patient died three hours postoperative.

He should have been operated upon earlier in life, as his abdomen was not sufficiently developed to contain the viscera removed from his chest.

Case 5.—N. G., female, age seven months, was admitted to the Babies Hospital, October 31, 1938, complaining of cyanotic attacks since birth. Roentgenograms showed left diaphragmatic hernia. Phrenic nerve was exposed in her neck two days before the hernia repair.

FIG. 2.—Hernia through the left foramen of Bochdalek before operation.

Operation.—This disclosed a left foramen of Bochdalek hernia. The left chest contained part of the stomach, most of the small intestine, the splenic flexure of the colon and the spleen. The defect in the diaphragm was closed with mattress sutures of black silk. Her convalescence was uneventful.

Follow-up: It is not 6.5 years postoperative, and the child is normal in every way.

Case 6.—V. D., male, age three weeks, was admitted to the Babies Hospital March 7, 1941. Weight 5,000 Gm. Cyanotic attacks since birth. Roentgenograms showed left

diaphragmatic hernia. The phrenic nerve was exposed in the neck two days before operation.

Operation.—A left foramen of Bochdalek hernia was found, with the left chest containing part of the stomach, several loops of small intestine, the colon as far as the splenic flexure and the spleen. The defect was repaired with mattress sutures of silk. Convalescence was uneventful.

Follow-up: Lost to follow-up after 1.5 years, at which time he was perfectly well and developing normally.

FIG. 3.—Same case three weeks after operation. All abdominal structures below the diaphragm.

Case 7.—J. B., female, age six days, was admitted to the Babies Hospital suffering from cyanotic episodes which were so severe that she was placed in oxygen 24 hours after birth. Roentgenograms showed a left diaphragmatic hernia.

Operation.—Age of 14 days: There was present an hernia through the left foramen of Bochdalek, with the left chest containing all of the small intestine, and the colon as far as the descending colon. The defect was closed with mattress sutures of black silk. Convalescence was uneventful. Discharged 19th day postoperative.

Follow-up: Three and one-half years later: She is well, and a roentgenogram of chest normal.

Case 8.—P. T., female, age 11 days, was admitted to the Babies Hospital suffering from cyanotic episodes since birth. Some attacks lasted several hours. Roentgenograms

of the chest showed left diaphragmatic hernia. The phrenic nerve was isolated two days before the repair of the hernia.

Operation.—Age of 22 days: Exploration showed that the left chest contained all of the small intestine, cecum and transverse colon through the left foramen of Bochdalek. The defect was closed with mattress sutures of silk. Convalescence was uneventful. Discharged 15 days postoperative.

Follow-up: Three years and nine months after operation: She is perfectly well. The roentgenogram of chest is normal.

Case 9.—D. K., male, age seven days, was admitted to the Babies Hospital with a history of severe cyanotic episodes since the fourth day of life. He had been kept in an oxygen tent most of the time since then. Vomited bile-stained fluid on several occasions in the past three days. A roentgenogram showed right diaphragmatic hernia. For the next ten days, he could not be taken out of oxygen long enough to be fed. After this, his condition improved considerably.

Operation.—Age three weeks, weight 2,550 Gm. Taken to the operating room in Davidson bed (oxygen), anesthetized and an intratracheal catheter inserted. At operation, a right foramen of Bochdalek hernia was found. The right chest contained all of the small intestine and the colon as far as the midtransverse colon. The defect was closed with mattress sutures of black silk. Convalescence was uneventful except for slight wound infection. Discharged on 21st day postoperative.

Follow-up: It is not 15 months postoperative; and he is in excellent condition, with no recurrence.

GROUP II—LARGE POSTERIOR DIAPHRAGMATIC DEFECTS

No Attachments of Posterior Diaphragm to Chest Wall, Deficiency of Diaphragmatic Leaf

Case 1.—A. E., male, age five months, was admitted to the Babies Hospital, March 13, 1934, with a history of dyspnea and cyanosis since birth. Roentgenologic examination showed small and large intestine herniated into the chest through a defect in the right diaphragm.

Operation.—The abdomen was opened through a right subcostal incision, and disclosed a defect in the posterior half of the right diaphragm, which corresponded to about one-half the size of the entire right leaf. Closure seemed impossible. The structures in the chest consisted of all of the small intestine beyond duodenojejunal junction; the cecum; the appendix and right half of the transverse colon; and the right kidney. All viscera were, however, reduced, and the defect closed with considerable difficulty. Convalescence was uneventful. The patient remained free from symptoms for about one and one-half years, when a routine roentgenogram of his chest, October 29, 1935, showed a recurrence.

Second Operation.—Age 2.5 years: Twenty-four hours before repair, under avertin anesthesia, the right phrenic nerve was exposed in the neck, a loop of plain gut placed about it for traction, so that it could be readily identified and crushed if necessary. Under ether anesthesia, 24 hours later, the scar of the right subcostal incision of the previous operation was excised, and the abdomen opened. Practically all of the structures that were in the chest at the time of the first operation had reherniated into it. There were many adhesions present, and reduction of contents necessitated considerable dissection. In order to close the defect, a right intercostal incision was made through the eighth space, which greatly facilitated the closure of the diaphragmatic defect. The phrenic nerve was then crushed from within the chest. Closure was accomplished with mattress sutures of black silk. The patient developed pneumonia and pleural effusion in the right chest postoperatively; 275 cc. of sterile fluid was removed from the right chest upon two occasions.

Follow-up: It is now eight years since the operation for recurrence of the hernia. He is entirely well, and the roentgenogram of the chest is normal.

Case 2.—P. J., female, age six days, was admitted to the Babies Hospital, April 25, 1944. She was one of twins, the daughter of a physician. Her twin brother was perfectly normal. The chief complaint was cyanosis and dyspnea since birth. First cyanotic attack occurred shortly after birth, and she seemed to become cyanotic every time her position was changed. A roentgenogram showed gas bubbles in the left chest.

Operation.—Age of 13 days; intratracheal anesthesia: There was a large defect in the posterior part of the left diaphragm with no attachment of the diaphragm to the chest wall. The left chest contained part of the stomach, all of the small intestine and the colon as far as the splenic flexure. The diaphragmatic defect was closed by passing silk mattress sutures out through the chest wall and tying them about the ribs. The only way the defect could be closed was to attach the diaphragm higher in the chest wall. Several interrupted silk sutures attached the edge of the diaphragm to the parietal pleura and intercostal muscle. Convalescence was uneventful. Discharged on the 18th postoperative day, weighing 200 Gm. more than her admission weight.

Follow-up: Admitted to the Babies Hospital three months after discharge with signs of intestinal obstruction. Roentgenograms showed no recurrence of the diaphragmatic hernia, but did show distended loops of jejunum. At operation, an obstruction was found in the upper jejunum due to adhesive bands. These were released, and her convalescence was quite uneventful. It is now more than one year since the closure of the diaphragmatic hernia and she is doing very well.

Case 3.—G. O., female, age two years, 11 months, was admitted to St. Luke's Hospital, May 24, 1937. She had not been well from birth, and was said to have always had lung trouble. Treated for pertussis for a long time because of cough. Diagnosis of pneumonia (unresolved), empyema, and later, "adult-type of Tb." Had had four months of bed rest. Her chest had been explored with needle 15 times. She had never had a chest roentgenogram.

Operation.—Large, left posterior diaphragmatic defect, with peritoneal sac, found. Her left chest contained stomach, all of the small intestine, the colon as far as the splenic flexure, and the spleen. The defect was closed with mattress sutures passed to the outside, with sutures of diaphragm higher in the chest wall. Convalescence was uneventful. Roentgenogram of chest seven years postoperative shows everything normal.

GROUP III—ESOPHAGEAL HIATUS HERNIA

Case 1.—L. Y., female, age six months, was admitted to the Babies Hospital, June 22, 1931, with a history of loss of weight and vomiting for three months. She was a pale, undernourished child, whose roentgenologic examination showed her stomach and transverse colon herniated into her chest through the esophageal hiatus. She continued to vomit while under observation for two weeks. The vomitus contained blood on several occasions.

Operation.—July 8, 1931: The abdomen was opened through a right subcostal incision. The entire stomach, the first and second portions of the duodenum, and almost all of the transverse colon were found to be in the chest, having herniated through a large esophageal hiatus. The viscera were reduced, and the esophageal hiatus was closed tightly around the esophagus after a large stomach tube had been passed. It was evident at operation that the esophagus was short, as it was only with considerable traction on the stomach that it could be kept in the abdomen. Convalescence was uneventful.

Follow-up: Roentgenologic examination, three months later, showed that a small portion of the stomach had reherniated into the chest. The patient has not been examined during the past year, but was doing well when last heard from.

This was about the result we expected in this case. At least operation kept duodenum and transverse colon from reëntering the chest.

GROUP IV—HERNIA THROUGH THE FORAMEN OF MORGAGNI

Case 1.—R. D., male, age 16 months. Weight 6,050 Gm. Shortness of breath since two months of age. Had always been a feeding problem. Mongolian idiot. Roentgenograms showed colon in the chest through the foramen of Morgagni.

Operation.—This showed presence of bilateral hernia through the foramen of Morgagni. Right hernia contained right lobe of liver, the ascending colon and half of the transverse colon. Left hernial sac was empty. Right hernia only was repaired. Died nine days postoperative from pneumonia.

Case 2.—J. V., male, age 12. Fatigue of one year's duration. Shortly after birth it was noticed that this child was mentally and physically retarded. Taken thyroid extract O. D. since infancy. Mental development approached normal but he was small for his age, very shy, and slow of speech. He was operated upon in another hospital two months ago for an acute appendicitis and a diaphragmatic hernia was discovered. B.M.R. was —28. Roentgenograms showed hernia through the foramen of Morgagni containing transverse colon.

Operation.—June 20, 1944: Bilateral hernia through the foramen of Morgagni. Peritoneal sacs removed and bilateral defects closed with mattress sutures of black silk. Convalescence was uneventful.

Follow-up: Entirely well 11 months postoperative.

GROUP V—EVENTRATION OF THE DIAPHRAGM

Case 1.—D. J., female, age one month. Normal delivery but was difficult to resuscitate, and was put in oxygen tent immediately after birth. Was too ill for the mother to see for the first four days of life. Discharged from the maternity hospital at age of ten days doing very well. Admitted to Babies Hospital with no symptoms. Roentgenologic diagnosis of diaphragmatic hernia made. After a period of convalesence during which she continued to gain weight she remained free from symptoms and was allowed home for a period of five weeks because she had blepharitis and conjunctivitis, and possibly a congenital heart.

Operation.—Age three months: Operation performed at Babies Hospital on October 20, 1941. Right subcostal incision disclosed eventration of the diaphragm. The right and left lobes of the liver as well as the stomach and part of the colon were above the normal diaphragmatic level. Only meager diaphragmatic muscle fibers were found and it was quite obvious that no surgical repair could be effected. It was also thought that the condition was compatible with life since there was no danger of obstruction of the structures. Discharged 13 days postoperative.

Follow-up: Developing normally, doing very well to date.

GROUP VI—CONGENITAL ABSENCE OF HEMIDIAPHRAGM

Case 1.—E. H., age three months; weight 4,230 Gm., was admitted to Babies Hospital with a history of cyanosis and dyspnea since birth. Normal spontaneous delivery, baby breathed normally. Noticed cyanosis next day. Roentgenograms upon admission showed abdominal structures in the left chest, and displacement of the heart.

Operation.—Left subcostal incision showed large stomach in left chest, spleen, jejunum, part of the left lobe of the liver also. No muscular diaphragm. Baby died three days after operation. Autopsy showed perforation of the stomach and necrosis of the left lobe of the liver. There was no muscular diaphragm but the left chest showed a large sac containing the stomach, jejunum, left lobe of the liver and the spleen. The perforation of the stomach was thought to be terminal, probably the result of volvulus. The baby also had hypertrophy of the myocardium, with a patent foramen ovale.

SUMMARY

1. Congenital diaphragmatic hernia occurs more often than was formerly suspected, and it should be ruled out in all cases showing obscure chest and upper abdominal symptoms.

2. The symptoms of congenital diaphragmatic hernia vary a great deal. They may be either cardiac, respiratory, gastro-intestinal, or a combination of all three.

TABLE I

TABULATION OF RESULTS IN 17 CASES

Male	Female	Age in Weeks	Cured	Died	Cause of Death
		Bochdalek Herniae (9)			
		Right—3			
2	1	5, 24, 1	2	1	Preoper. intestinal obst.
		Left—6			
2	3	18, 2, 28, 8, 1	5		
		Also Defect in Dome (1)			
1		9 years		1	Shock
		Large Posterior Defects			
		Right (1)		Left (2)	
1		20 weeks	1 (recurrence)		Cured
	1	1 week	1		
	1	3 years	1		

OTHER TYPES

Esophageal hiatus:	Female	6 mos.	Cured	Minimal recurrence
Morgagni:	Male	16 mos.	Died	Postoper. pneumonia (Mongolian idiot)
	Male	12 yrs.	Cured	
Eventration:	Female	3 mos.	Surviving	
Congenital absence:	Male	3 mos.	Died	Perforation of stomach. Liver necrosis

3. The diagnosis may be made by a roentgenogram of the chest, which shows gas bubbles in the place of normal lung, collapsed lung and displacement of the heart away from the affected side.

4. Physical signs in the chest are often so confusing that the diagnosis may be missed if a roentgenogram is not taken.

5. Surgical repair of the hernia should be advised immediately for all cases where either small or large intestine is in the chest because of the danger of intestinal obstruction.

6. The author prefers the abdominal approach through a subcostal incision, with exposure of the phrenic nerve in the neck 48 hours before repair. Many surgeons prefer the thoracic approach.

7. Positive pressure or intratracheal anesthesia are very desirable.

8. All patients should be kept in an oxygen tent for several days after operation.

9. Fluid requirements should be maintained after operation.

10. Pleural effusion may occur, and the chest should be aspirated as is necessary.

11. Seventeen cases of congenital diaphragmatic hernia which were operated upon have been reported.

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IDIOPATHIC DILATATION OF THE ESOPHAGUS*

DIFFERENTIATION OF CLINICAL TYPES AND SUCCESSFUL OPERATION IN INTRACTABLE CASES

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Today it is recognized that dilatation of the esophagus without obvious pathologic cause is a common condition. In fact, it has approximately one-third the frequency of carcinoma of the esophagus, being second only to the latter in the statistics of the larger clinics (MacMillan[1]). It appeared earlier in the literature under the purely descriptive terms, ectasia or idiopathic dilatation of the esophagus. But today the author's etiologic conception is usually introduced into the name and this condition is most commonly designated as achalasia of the cardia or as cardiospasm, though there are still widely conflicting opinions about the cause of the dilatation. Fortunately for the patients who suffer from this serious, and usually progressive, difficulty in the passage of food from the dilated esophagus into the stomach, treatment does not need to wait for a final determination of cause; by dilating the cardia, either on one or repeated occasions, the symptoms in the majority of cases are adequately controlled. However, some of these patients do not respond satisfactorily to this and the other conservative measures of treatment. Although the percentage of this refractory group is small this failure to respond to conservative measures offers a serious problem for two reasons: First, because of the complete failure of the usual therapeutic measures to alleviate the condition; and, secondly, because of the progressive seriousness of their symptoms. They constitute a small but important group of cases, and their relief has become a challenge to the general surgeon as well as to the esophagoscopist.

In studying the problem presented by these cases of idiopathic dilatation of the esophagus not adequately controlled by conservative measures, certain observations have been made that I believe help: (1) To clarify our understanding of the mechanism involved; (2) to subdivide these resistant cases into clinical types which show important physiologic and probably etiologic differences; and (3) to attack the difficult therapeutic problem involved.

The first clinical description of idiopathic dilatation of the esophagus was found by Dr. Ralph Major,[2] and included in his collected classic descriptions of disease. This case was described by Thomas Willis in his Pharmaceutica Rationalis, published in London, in 1674. This great seventeenth century physician was, like his contemporary Thomas Sydenham, an accurate and

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painstaking observer of clinical manifestations. Not only did he give an excellent description of the characteristic symptoms, but in addition to this he established the principle of treatment by instrumental dilatation, and even forecast the persisting conflict of opinion as to whether or not the primary cause is a disturbance of the extrinsic nervous mechanism in the region of the cardia. Little was added to our knowledge concerning this condition for another two hundred years, during the latter half century of which isolated cases were reported as pathologic and clinical oddities. Thus, in 1876, Zenker and Ziemssen[3] were able to collect only 18 cases, not including Willis's case, of 1674, and a case particularly well described by Purton,[4] in 1821. It is to the clinical work of Mikulicz[5] and the experimental work of Meltzer[6] that we owe the conception and even the name of cardiospasm as the first major step toward establishing the mechanism and etiology of the dilatation. This theory had a rather general acceptance until certain serious objections were raised to it. To Hurst[7] we owe chiefly the hypothesis that the difficulty in the passage of food from the dilated esophagus into the stomach is not due to spasm but is caused by the failure of relaxation to occur in the terminal portion of the esophagus when the advancing peristaltic wave reaches it. In conformity with this conception he coined the term "achalasia" of the cardia. Hurst felt that the ease with which dilators passed through the esophagus into the stomach was incompatible with the conception of spasm of the cardia, as was also the absence of hypertrophy of the circular muscle at this point upon necropsy. Another difficulty that is faced by any theory based on abnormality of the nervous mechanism of the cardia, whether cardiospasm or achalasia, is to explain why the dilatation of the esophagus usually ends not at the cardia but above that point, approximately at the level of the diaphragmatic hiatus; also why, in the human, no important condensation of the circular muscle acting as an anatomic sphincter can be found at the cardia. The best answer to these objections is found in the experimental production of cardiospasm by cutting both vagus nerves in animals.[8, 9] This demonstrates the presence of a broad physiologic sphincter sufficiently powerful not only to produce the typical dilatation of the lower esophagus but actually to cause the death of the animal from failure of food to pass into the stomach. The theory of achalasia has been considerably bolstered by microscopic findings of pathologic changes in the myenteric plexus at the lower end of the esophagus. Rake[10] first convincingly demonstrated these lesions, and they have been confirmed by other observers,[11] although there are some typical cases that have failed to show such changes.[12] (A possible explanation of these exceptions is that the involvement of the vagus tract may be at any point from the brain to the terminal nerve plate.) Although it has not been definitely proved that such degeneration of the ganglion cells in Auerbach's plexus is primary rather than secondary, probably this theory of achalasia of the cardia is today the most widely accepted hypothesis of the etiology of idiopathic dilatation of the esophagus. However, there are important opinions still adverse to it.

Jackson,[13] on the basis of a very extensive observation of these cases through the esophagoscope, believes that the obstruction to the esophagus is due to phrenospasm, while Mosher[14] feels that the cause is a peri-esophageal fibrositis at the hiatal portion of the esophagus. An excellent review of the literature to that date was presented by Sturtevant[15] in 1933.

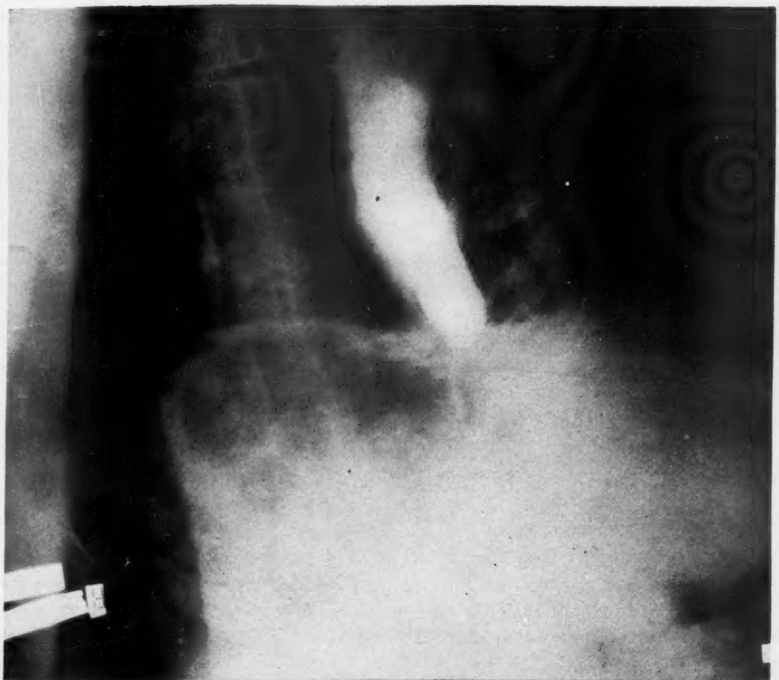
This, in brief, is the present status of our knowledge concerning the etiology of idiopathic dilatation of the esophagus. Nearly all of the investigators who have contributed recent studies of the condition have assumed that the clinical syndrome is regularly due to some one fundamental cause, though they have varied among themselves as to what this cause is. If other physical conditions appeared to operate in individual cases, then they were considered to be secondary factors which had arisen to complicate the picture during the lengthy course that is usually characteristic of this condition. As the result of observations made in the cases cited below, correlated with certain facts already recorded in various places in the literature, we believe that such an assumption of a single basic etiology is fallacious and that the characteristic clinical syndrome is caused by several entirely different primary causes. It is important to differentiate these various fundamental types, both to obtain a better understanding of this frequent disease, and also for the practical assistance that it affords in the management of the intractable group.

CLINICAL OBSERVATIONS

Hurst's conception of an achalasia of the cardial end of the esophagus due to some abnormality in its vagal innervation is accepted in this study as the most plausible explanation in the majority of cases for the esophageal dilatation and for the difficulty that is observed in the passage of material into the stomach. Our first case to throw doubt on the universal applicability of this theory was the following:

Case 1.—C. K., a 65-year-old paperhanger, was admitted to the hospital, September 15, 1927, on account of dysphagia and regurgitant vomiting associated with dull aching distress of three months' duration. Even the swallowing of water may now cause regurgitation. The dysphagia increased in severity and he had lost 22 pounds in weight in the last three months. Fifteen months before the onset of these persistent symptoms, he had had an attack of sharp noncharacteristic epigastric pain radiating around both sides to the back and lasting three weeks. The only physical findings of significance on examination were evidence of malnutrition with recent weight loss and moderate dehydration. His blood picture was that of a moderate secondary anemia. His roentgenologic examination showed a typical moderate dilatation of the esophagus with peristaltic waves and ending in a smooth cone at approximately the level of the diaphragm (Fig. 1). Esophagoscopic examination verified the dilatation of the esophagus, but due to the fact that only the short esophagoscope was being used, the terminal cone of the esophagus was not visualized at this time. In view of the patient's emaciation, a gastrostomy tube was placed in the stomach transabdominally. The operative incision was so short that no exploration was feasible, except that no gross malignancy of the stomach was felt on palpation with one finger. Feedings through the gastrostomy tube quickly rectified the patient's nutritional deficiency when repeated roentgenologic examinations and esophagoscopy (with 53-cm. scope) verified the diagnosis of dilatation of the esophagus without obvious cause. Repeated dilatation of the cardia (with the

FIG. 1



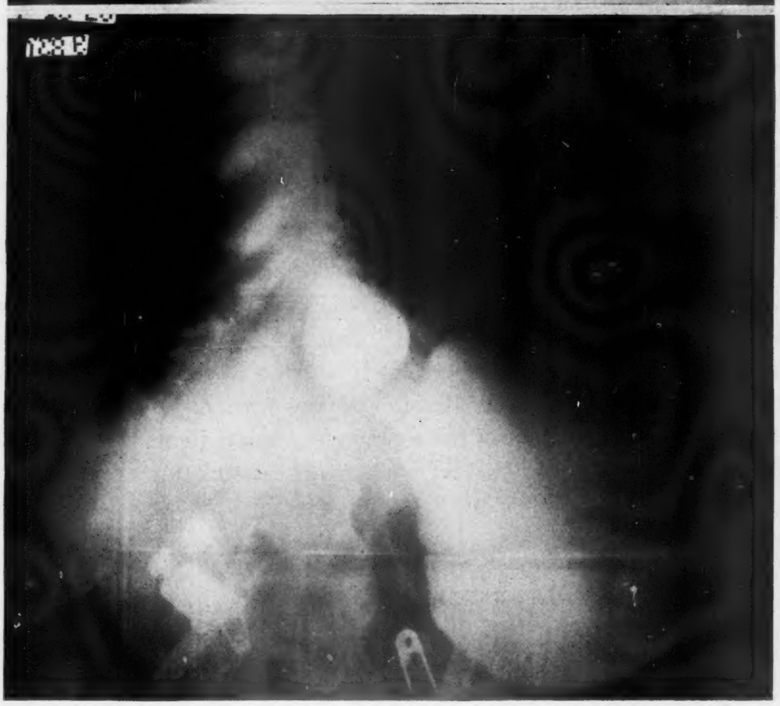


FIG. 2

FIG. 1—Case 1: Dilatation of the esophagus found on first examination August 1, 1927, proven later to be cardiospasm apparently reflex from an ulcer of the stomach.

FIG. 2.—Case 1: Showing dilatation of the esophagus still present ten months later but now also accompanied by a pylorospasm, patient being fed entirely by gastrostomy tube.

large-size Plummer dilator) caused temporary improvement in the ability to swallow food, which benefit, however, lasted only for a few days each time, and the dysphagia always returned. On this account, the gastrostomy opening was not allowed to close and most of the feeding had to be given by tube. About ten months after the gastrostomy was performed, he developed severe pain in the epigastrium after tube feedings, accompanied by huge peristaltic waves in the stomach. Roentgenologic examination of the stomach merely verified the delay in its emptying without demonstrating any cause for this. It was obvious that the patient now had a pyloric obstruction or spasm in addition to the persistent cardiospasm (Fig. 2). The symptoms of both continued with periods of exacerbation and remission for another ten months when the pain and difficulty in giving sufficient food either by mouth or by tube necessitated an abdominal exploration. An ulcer of the stomach was found on the lesser curvature of the stomach a little beyond the incisura angularis. There was no obstruction either at the cardia or the pylorus. A subtotal gastrectomy was carried out, including about two-thirds of the lesser curvature. The antecolic anastomosis between the end of the stomach and the side of the jejunum (Pólya-Moynihan type) did not come near to the cardia. Almost immediately after operation the patient's ability to swallow food by mouth began to improve so that by the seventh postoperative day, he was taking solid foods without difficulty and the gastrostomy tube was removed. The gastrostomy opening healed slowly (it had been maintained for 21 months) and during the time it remained open he still had some dysphagia.

Subsequent Course: Since the closure of the gastrostomy fistula (now 11 years) the patient has not had any further major difficulty in the swallowing of his food. The eating of very dry food occasionally causes a sticking sensation and he has, on a few occasions, regurgitated a mouthful or so of food. He is particularly apt to be quite constipated at such times. Two months after gastrectomy roentgenologic examination showed "on ingestion of liquid barium suspension there was scarcely any delay in the lower esophagus. The barium passed through the cardiac sphincter in a more or less continuous stream. The marked obstruction previously noted had disappeared and, at the most, there was only a very slight temporary delay. On ingestion of barium cereal paste the delay was more pronounced, though it passed through without being regurgitated." And two years following gastrectomy roentgenologic examination of the esophagus showed no evidence of dilatation and no delay in the passage of the barium mixture into the stomach.

COMMENT: In this case, the syndrome of idiopathic dilatation of the esophagus was present with its well-marked symptomatology and the characteristic roentgenographic appearance. This syndrome disappeared after the resection of a gastric ulcer. It is extremely difficult to explain such a sequence of events by the hypothesis of achalasia (as opposed to cardiospasm). It seems quite clear that the dilatation of the esophagus occurred chiefly in response to a reflex disturbance (presumably sympathetic stimulation), which in this case caused spasm of the cardia first and later also an associated spasm of the pylorus.

This case with its lasting relief following removal of the focus of reflex irritability suggested that possibly information of value might be obtained by temporary paralysis of the sympathetic innervation to the cardia while the vagus innervation is not disturbed. It was hoped that we might be able to achieve this result by spinal anesthesia, just as we have previously used this procedure to subdivide cases of megacolon.[16] The efferent sympathetic

pathways to the cardia seem to be very complex[17] and there is no exact knowledge of the precise levels at which they leave the cord. Therefore, when this investigation was begun it was uncertain whether or not anesthesia would be a helpful tool of investigation in it. The following case is the first one that presented itself to us to determine this point:

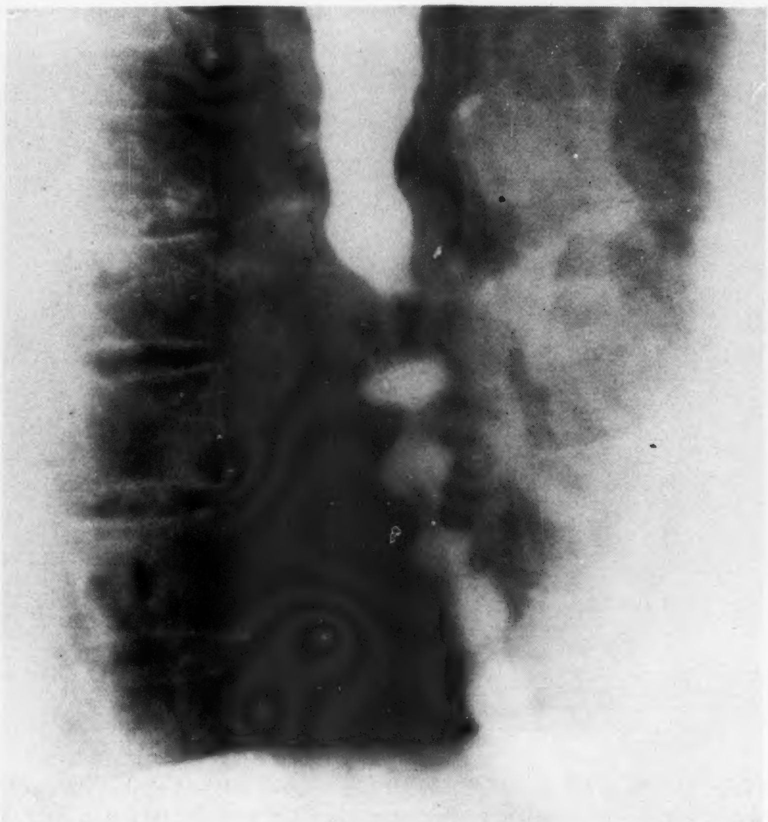


FIG. 3.—Case 2: Spasm of the lower third of the esophagus.

Case 2.—H. G., a 56-year-old man, came into the hospital in February, 1929, on account of dysphagia. His symptoms came on rather suddenly two years ago when he had difficulty in swallowing a piece of bread; it seemed to him as if something closed in his throat and he regurgitated the bread. Since then he has had similar difficulty in swallowing masticated solid foods unless he "washes them down" with *warm* fluids. Even with this assistance it seems to stop for an interval near the stomach before it feels as if it goes into this viscus. Sometimes even with the regimen of slow eating followed by warm fluid he has to vomit because the food will not pass. He can drink hot liquids readily but he has not been able to tolerate cold water since the onset of his illness. It feels as if the latter "stops right in the pit of the stomach" and then he regurgitates the water. The dysphagia has been progressive, particularly in the last few months when he has been having great difficulty in getting the food into the

stomach, during which time he breaks out into a hot sweat and following the lengthened meal he feels exhausted. He has lost 20 pounds during the course of the illness but seems to be maintaining his weight at present. One year previously he was examined by Dr. Chevalier Jackson who dilated his lower esophagus. Following this his symptoms were much less marked but this improvement lasted for only two weeks. Two other dilatations were followed by very little benefit. Medication with belladonna was also ineffective. In his past history he had two attacks of acute epigastric pain which suggested gallbladder disease to several physicians but a cholecystogram did not afford

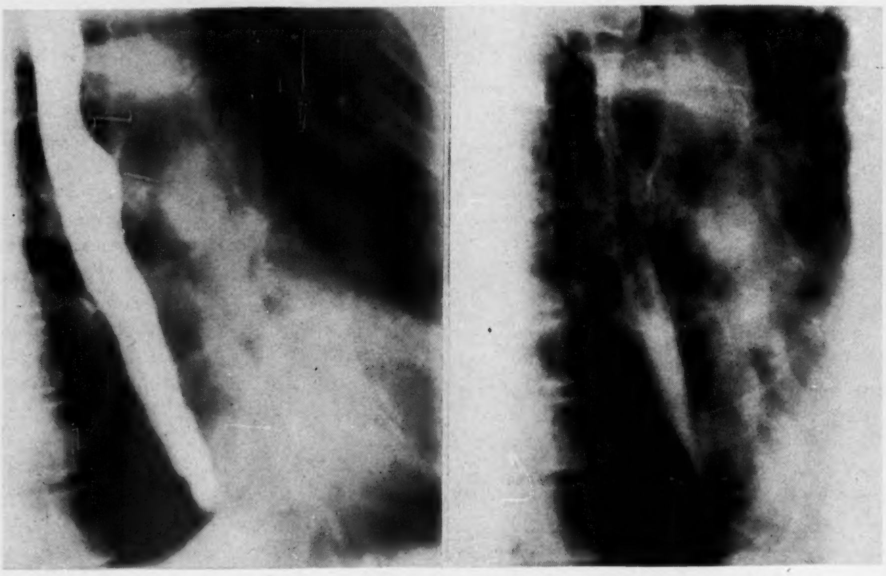


FIG. 4A FIG. 4B

FIG. 4.—Case 2: Release of spasm of the lower third of the esophagus by spinal anesthesia (A) 25 minutes after intraspinal novocaine injection, immediately after ingestion of barium. (B) 30 minutes after intraspinal novocaine injection and five minutes after ingestion of barium. The spasm in the lower third of the esophagus has completely disappeared after the induction of spinal anesthesia.

any definite corroboration of such, although one surgeon advised cholecystectomy. Also, he had two attacks of pain suggestive of renal colic with hematuria a year ago but investigation of his upper urinary tract by a competent urologist failed to disclose any definite pathology there and no stones were visualized roentgenologically. These symptoms have not recurred.

Physical examination revealed a moderate hypertension with possibly a slight enlargement of the heart to the left. There was no particular tenderness at any point in the abdomen. The Wassermann reaction in the blood was negative.

Roentgenologic examination revealed no abnormality in the passage of the barium mixture through the upper esophagus, but the lower third of the esophagus showed multiple gross areas of spastic contraction which held the barium in segmental accumulations and greatly retarded its progress through this region. There were at least seven or eight constrictions in the lower esophagus though their configuration was constantly changing (Fig. 3). When the barium got through this contorted portion in the lower esophagus it passed through the cardia into the stomach without further delay.

Under fluoroscopic control, a Plummer bag was drawn down into the area of spasm along a silk thread, and expanded to a water pressure of 18 to 20 pounds for five minutes without discomfort. This dilatation was repeated twice more at weekly intervals,

but as with the previous course of dilatations the improvement in symptoms was only ephemeral.

The effect of various drugs and spinal anesthesia on the spasm of the lower esophagus was then studied. Atropine and ephedrine produced no striking change in the spasm and intensive treatment with tincture of belladonna produced no improvement in symptoms. On the other hand, the effect of spinal anesthesia was very striking. Anesthesia was obtained to the level of T-8 and well-marked hypo-esthesia to T-4.

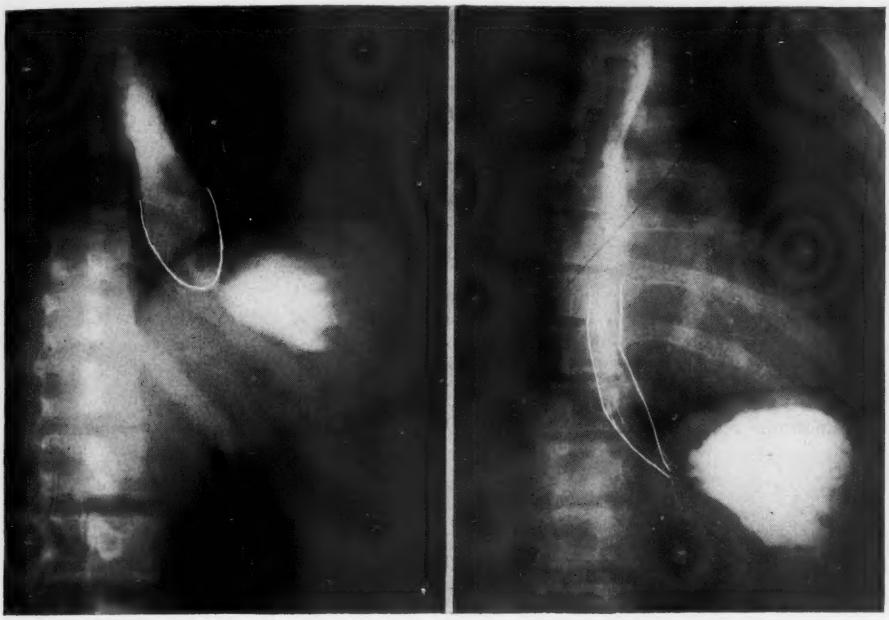


FIG. 5 FIG. 6

FIG. 5.—Case 3: Dilatation of the esophagus control film.
FIG. 6.—Case 3: Dilatation of the esophagus entirely disappears during spinal anesthesia.

There was no marked fall in blood pressure. During the height of the anesthesia the spasm of the lower esophagus disappeared almost completely and the delay in the emptying of the esophagus into the stomach was greatly diminished (Fig. 4).

COMMENT: It had been hoped that the temporary interruption of this spasm in the lower esophagus might lead to a gradual improvement in symptoms as we have succeeded in obtaining in instances of Hirschsprung's disease.[18] However, the patient was lost track of soon after this and we do not know whether there was any therapeutic influence of the spinal anesthesia on the later course of the disease. Telford and Simmons[19] have reported one case with an excellent clinical result following a spinal anesthesia.

The result in this first case, although it was one of persistent spasm involving the lower third of the esophagus (like those reported by Schmidt[20]) and not a typical one of idiopathic dilatation, led us to believe that we could achieve by this means a satisfactory paralysis of the sympathetic nerves to the lower esophagus. We, therefore, used spinal anesthesia as a method of

investigation in typical instances of idiopathic dilatation of the esophagus and found that such cases could be divided into two groups: (A) Those that respond by temporary relief of the dilatation; and (B) those that show no marked improvement after satisfactory spinal anesthesia to the level of the sixth thoracic segment. The following case is a typical example of Group A:

Case 3.—J. P., a 36-year-old factory worker came into the hospital on account of dysphagia of three months' duration. He first noted a sticking sensation on eating solid food, often associated with moderately sharp, somewhat crampy pain. On account of this difficulty in swallowing solid foods he has had to confine himself practically to a liquid diet, and he has lost 25 pounds in weight. Liquids usually pass quite freely if he drinks slowly. *Past History:* During the last year he has had a few attacks of sharp epigastric pain radiating to the right shoulder. In the present illness this radiation seems to have been associated with the difficulty in swallowing. Also, for the past two years he has had some frequency of bowel movement with mucous shreds.

Physical examination was essentially negative throughout, except for evidence of moderate weight loss. The patient is of a rather nervous temperament. Routine laboratory studies were negative, except for a positive guaiac reaction in a liquid stool. Roentgenologic examination showed a fusiform dilatation of the lower esophagus with a marked delay in the passage of barium from the lower part of the cone which was perfectly smooth (Fig. 5). There was some irregularity in the filling of the duodenal cap. However, it was difficult to get enough barium into the stomach for satisfactory examination. An oral cholecystogram showed an agglutination of small calcified masses in the fundus of the gallbladder, with good concentration of the dye and normal emptying. Esophagoscopic examination showed considerable retention in a moderately dilated esophagus without much maceration of the mucous membrane. Just below the level of the diaphragm there was a small superficial ulceration noted on the posterior wall and there was some reddening of the mucous membrane. Just below this the lumen was reduced to a small opening which failed to relax with bougie dilatation so that only a small tip could be forced into the stomach. There was no evidence of malignancy. Spinal anesthesia was carried out, which was complete to T-7. Immediately after anesthesia was attained the dilatation and stasis in the esophagus entirely disappeared and the barium mixture passed without delay into the stomach (Fig. 6). A diagnosis of cardiospasm, probably secondary to cholelithiasis was made, and the patient was explored. In addition to multiple amorphous stones in the gallbladder the patient had an ulcerated lesion on the midposterior wall of the stomach suggesting carcinoma, with extensions involving the posterior peritoneum above the pancreas. The cardia of the stomach itself was not grossly involved. Due to the posterior peritoneal involvement, however, the lesion was inoperable.

COMMENT: In this case spinal anesthesia demonstrated that dilatation of the lower esophagus and the dysphagia which it produces can be temporarily overcome in certain instances by paralysis of the sympathetic innervation to the level of the sixth thoracic segment. The following two cases, however, show instances of typical idiopathic dilatation of the esophagus where satisfactory spinal anesthesia does not produce any striking change in the condition:

Case 4.—F. S., a 25-year-old housewife, came into the hospital, September 29, 1938, on account of dysphagia. The difficulty in swallowing began rather gradually about seven months previously, and has been increasing since that time. In attempting to swallow she has a painful, choking feeling and then regurgitates her food, which relieves this sensation. At times she is even unable to keep water down. For a time after the

FIG. 7

FIG. 8

FIG. 9

FIG. 7.—Case 4: Dilatation of the esophagus control film.
FIG. 8.—Case 4: Dilatation of the esophagus not relieved by satisfactory spinal anesthesia.
FIG. 9.—Case 4: Postoperative result. Dilatation of the esophagus has been entirely relieved by esophagogastrostomy.

onset of the symptoms she thought by taking soft solids that she was able to swallow more readily. Now, however, she thinks that it makes little difference what she eats. She is constantly regurgitating both solid food and liquids. There is some fluctuation in the severity of the dysphagia. She has lost 17 pounds from her original weight of 110 pounds. Beginning two months after the onset of her symptoms she had several esophageal dilatations. The first of these afforded her a considerable relief of symptoms which lasted, however, for only a few days and the subsequent dilatations have been even less effective. She is thoroughly discouraged by the difficulty that she has in swallowing food and by its apparent progressive course, recently having more trouble in even getting liquids down. Her past history and system review appears to be entirely negative as far as relevant data is concerned. She had uncomplicated pneumonia at the age of 15.

Her physical examination shows evidence of recent weight loss but otherwise is negative. Routine laboratory studies, including blood Wassermann, are negative. Roentgenologic examination showed a typical, well-marked dilatation of the esophagus ending in a smooth cone at approximately the level of the diaphragm (Fig. 7). Esophagoscopy had to be carried out under general anesthesia as the patient refused to allow it under local. The esophagus showed marked dilatation even of its upper portion and it contained much retained food and mucus. After evacuation of its contents the lower end was examined. The dilated cone ended in a constricted area which would allow only a rather small esophageal bougie to pass. It was dilated as well as possible with bougies as the Plummer bag would not pass the contracted zone. Considerable relief was obtained following this dilatation, which improvement lasted, however, for only three days. After the initial dilatation this patient had six further dilatations, the dilatation being carried out to the point where at one time the esophagoscope could be passed into the stomach. After each dilatation she had temporary improvement which disappeared within only a few days, and it was impossible to make the patient gain weight. Spinal anesthesia was given on two occasions as a test of motor function. At the first time the level of anesthesia was unsatisfactory, complete anesthesia being obtained only to about T-11. At the second time, however, anesthesia was obtained to the level of T-6 and hypo-esthesia two or three segments above this point. In spite of an entirely satisfactory anesthesia the dilatation of the esophagus remained practically unaltered (Fig. 8). It was thought that there might be possibly a slight improvement in the passage of barium into the stomach. This, however, was not striking. The test was interpreted as showing that satisfactory spinal anesthesia in this instance did not materially influence the dilatation and the stasis in the esophagus.

Case 5.—E. P., a 78-year-old retired nurse, came into the hospital, May 25, 1939, on account of dysphagia and vomiting of five months' duration. A short time before the onset of these symptoms she had contracted a cold, associated with a sense of tightness in her chest, which kept her in bed for ten days. Since then she has had a persistent, rather unproductive cough and has felt fatigued. The difficulty in keeping food and vomiting began at that time and has progressed since then so that she now vomits a considerable part of the food she takes. About three weeks prior to admission she began having severe paroxysms of substernal distress relieved only by vomiting. She has identified unaltered food eaten as long as 24 hours previously. She has lost 20 pounds in weight during the present illness. Eight years ago the patient had severe pains in the chest during meals, accompanied by a feeling that the food did not pass freely into the stomach. These attacks occurred two or three times a week and persisted in a modified form until two years ago when they disappeared after taking dilute hydrochloric acid with every meal.

Past History: She has had some palpitation of the heart with orthopnea for ten years, for which her physician has given her digitalis. Fifteen years ago she had a bleeding duodenal ulcer, for which a gastro-enterostomy was carried out together with the removal of a gallbladder containing stones.

On physical examination the patient was a well-preserved woman for her age, but she showed evidence of rather marked weight loss. There were fine râles at both lung bases. A soft, blowing systolic murmur at the apex of the heart. The abdomen contained no masses and there was no tenderness. Wassermann reaction in the blood was negative. There was albumin without casts in the urine, and on admission a white count of 18,000, with 77 per cent polymorphonuclear leukocytes. The Wassermann

FIG. 10.—Case 5: Dilatation of the esophagus with compound "S"-shaped curve and fluid level in the upper portion of the "S." This is idiopathic dilatation of the esophagus of the dolicho-esophagus type.

reaction in the formed stool was negative. Roentgenologic examination showed a greatly dilated and very tortuous esophagus in the shape of an "S" with the upper pouch extending to the right of the midline. The esophagus then rises toward the left, makes a hairpin turn and comes down again just to the right of the midline. The dilated portion ends perfectly smoothly at approximately the level of the diaphragmatic hiatus (Fig. 10).

The major portion of the barium mixture given was still in the esophagus at the end of six hours. Esophagoscopy showed marked retention of cloudy fluid and food particles with a typically pasty-looking mucous membrane at the level of the diaphragm. The mucous membrane folds lie in apposition to each other but offer no resistance to the passage of the esophagoscope through the cardia. Three dilatations of the cardia gave little or no relief. In spite of this treatment combined with other conservative measures, such as esophageal lavage, atropine, *etc.*, it was impossible to get sufficient nourishment for her. A satisfactory spinal anesthesia was given (complete to the level of T-7) without marked benefit either to the patient's ability to swallow or to the dilatation of the esophagus. Possibly barium got through from the lower pouch into the stomach slightly more easily.

COMMENT: In Cases 4 and 5, then, satisfactory spinal anesthesia produced no striking change in two typical examples of idiopathic dilatation of the esophagus. These, therefore, fall into our Group B, in which sympathetic overactivity apparently plays no rôle in the production of the syndrome, while Cases 1, 2, and 3 fall into Group A, where the esophageal abnormality is apparently on the basis of sympathetic overactivity. Unfortunately, we have not yet had an opportunity to examine the reaction to spinal anesthesia in simple cases of idiopathic dilatation of the esophagus which respond readily to dilatation.

CLINICAL TYPES

From an analysis of our series of 85 cases of idiopathic dilatation of the esophagus (usually diagnosed as achalasia of the cardia or as cardiospasm), and aided by the information obtained from the use of spinal anesthesia as a tool of investigation, we feel that it is now possible to recognize four different clinical types which probably also differ from each other in their etiology. These types are: (1) Achalasia of the esophagus; (2) true cardiospasm; (3) dilatation associated with a constriction at, or just above, the cardia; and (4) dolicho-esophagus. Let us investigate separately each of these clinical groups.

1. *Achalasia of the esophagus* is probably the most common variety encountered. It usually readily responds to dilatation of the esophagus and is due to an incoördination of the neuromuscular apparatus at the lower end of the esophagus, whereby the cardia fails to relax properly. At present, there are no pathognomonic criteria clinically of such a failure of relaxation and, consequently, the diagnosis of this type must be made largely by exclusion of the other three types, though probably most of the cases of idiopathic dilatation of the esophagus that respond promptly to simple dilatation and whose symptoms are readily controlled by this method fall in this category.

2. *True Cardiospasm:* The roentgenographic appearance in this clinical type appears identical to that seen in achalasia. However, in this clinical syndrome, a reflex focus of irritation can be found, the eradication of which will largely or completely remove the difficulty in the passage of food from the esophagus into the stomach. Such cases respond to spinal anesthesia with temporary relief. During the anesthesia, the free passage of barium

into the stomach can be seen under the fluoroscope. Probably the most frequent foci for the origin of the reflex causing the cardiospasm are ulcer of the stomach or duodenum, other irritated lesions involving the posterior peritoneum, and cholelithiasis.

3. *Dilatation Associated with Constriction at the Cardia:* This group probably represents a small percentage of the cases of idiopathic dilatation of the esophagus. Our fourth case, however, seems to be definitely a member of this group. At operation, a circular fibromuscular band was found around the region of the cardia and the lumen of the latter would not admit a lead pencil. There was no evidence of any recent acute inflammatory reaction associated with this fibrosis. Whether this was fibrositis due to periesophagitis as Mosier hypothecates or whether a congenital abnormality somewhat like hypertrophic pyloric stenosis with symptoms showing, for some unexplained reason, only at a later date cannot be decided from the evidence at hand. There are, in the literature, however, well-authenticated cases in which a thickening in the neighborhood of the cardia is found of sufficient degree so that even at postmortem examination the dilated esophagus filled with fluid will hold its contents, while the common type of dilatation of the esophagus empties itself readily.[21] Knight,[22] Case 3, described as an hypertrophy of the cardiac sphincter, is a typical example of this group. Consequently, it seems to us unquestionable that such constricting lesion of the lower esophagus or cardia occurs as the cause of a definite but small group of cases of dilatation of the esophagus without clinically discoverable cause. Spinal anesthesia will, of course, not relax such a lesion as was proven in our Case 4, and dilatation *per os* will probably prove to give only a relatively short period of relief of symptoms, as was also true in this case. The final diagnosis of this type will probably require identification by proving the presence of the fibromuscular ring.

4. *Dolicho-esophagus:* It is generally recognized by all who have clinical experience with idiopathic dilatation of the esophagus that the esophagus is usually also longer than normal. This is easily recognized in esophagoscoping such cases, and such a moderate increase in the length of the esophagus is not meant in the classification of the group of dolicho-esophagus. However, there is a group, a characteristic example of which is our Case 5, in which there is so extreme an increase in the length of the esophagus that the latter forms a compound curve and assumes an "S"-shape. Most observers have assumed that this extreme lengthening of the esophagus is due to the same cause as the lengthening previously mentioned as associated with practically all cases of idiopathic dilatation, namely, the hydrostatic pressure of the column of fluid in the dilated esophagus. Two facts make such an explanation highly improbable. In the first place, the form of the curve is "S"-shaped with one pocket at a much higher level than the other one. This is quite a constant finding and cannot be explained on the basis of hydrostatic pressure which would tend to produce the usual fusiform or pyriform shape

seen in the common example of achalasia. In the second place, the combination of such a lengthening of the esophagus associated with marked dilatation has been found in infancy.[23] It seems much more probable that this is an abnormality of the esophagus comparable to the dolichocolon, in which there is a congenital increase in length which is responsible for the compound curving of the structure and also causes an hydrostatic functional obstruction due to kinking of the lower end when the dilated and distorted esophagus is filled with fluid. We do not believe that this form of extreme lengthening of the esophagus in an "S"-shaped form is merely secondary to some disturbance in the emptying of the esophagus into the cardia, but we rather believe that it is the primary cause in these instances of the functional obstruction. An esophagoscope can be readily passed in these cases through the cardia, which is patent, into the stomach when it can be safely gotten down past the "S"-shaped curve of the esophagus. This was done in our case number five and the obstruction seems to be due merely to the functional condition present. In this type the dilated esophagus acts like a sac filled with fluid and obstructs the cardia by partially kinking the esophagus at the diaphragm. Another piece of evidence supporting this explanation for the dolicho-esophagus group is seen in the experience in such a case reported by Freeman[24] in 1923. Twenty years previously he had carried out an esophagoplasty in the neck which had shortened the esophagus. This procedure caused immediate relief of symptoms which continued for 20 years.

TREATMENT OF INTRACTABLE CASES

Everyone is agreed that the treatment for the majority of cases of idiopathic dilatation of the esophagus is relatively simple and effective. Dilatation of the esophagus produces in most cases a marked relief of symptoms, which occasionally is permanent but more often recurs after a variable interval. However, by repeated dilatations at regular or irregular intervals, the great majority of patients have sufficient relief, so that no more drastic method of attack is required. In a small group, however, such dilatation and all other conservative measures fail to produce any lasting improvement. The symptoms in such cases tend to become more severe and the problem of the patient's getting sufficient nourishment becomes a serious one. It is in this group of cases, intractable to dilatation and other conservative measures, that operative relief should be considered. Obviously, in the severe cases of true cardiospasm the focus of irritation should be removed. When this is accomplished, as in Case 1, the esophageal symptoms practically disappear, although some degree of dilatation of the esophagus is likely to remain for many years, as is the case in its counterpart, Hirschsprung's disease of the colon. When, however, the idiopathic dilatation of the esophagus is not due to a reflex irritation of the sympathetic innervation (as proven, for example, by spinal anesthesia) and relief is not obtained by dilatation and other conservative measures, then relief of the condition by operation is indicated if

the symptoms are sufficiently urgent or the course is progressively downward. Usually a tube can be gotten down into the stomach so that gastrostomy as a preparatory measure is not necessary.

An excellent review of the various operations undertaken in these resistant cases, with the results attained, is given by Ochsner and DeBakey.[25] There have, in general, been four different types of operation advocated for the relief of the esophageal stasis. Historically, the first operation is that devised by Mikulicz, in which the cardia of the stomach is mechanically dilated transgastrically at celiotomy. The rational of this procedure was that manual dilatation produced a much greater degree of stretching of the cardia than can be produced instrumentally through the esophagus. Its disadvantages were two-fold: One, the danger of tearing the esophagus during the process of dilatation; and, secondly, the return of symptoms after the recovery by the cardia from the dilatation. It is still occasionally employed today, though it is usually regarded as inadequate.

A second type of operative attack is that advocated by Heller. In this procedure, the outer fibromuscular coats of the esophagus are divided longitudinally through the region of the cardia down to the submucosa. This is comparable to the very satisfactory operation employed to relieve congenital hypertrophic pyloric stenosis. Theoretically, this operation would be effective chiefly where there was an hypertrophic fibromuscular band, as for example in our third clinical group, but in the other types it would be relatively ineffective. As a matter of experience, it has been found that there is a high incidence of recurrence of symptoms after this operation.

A third operative approach, recently devised, was an effort to eliminate the sympathetic supply to the cardia. This attempt was based primarily upon experimental work in the laboratory, where it was found that the dilatation of the esophagus following section of the vagus nerves was prevented, if the sympathetic innervation of the cardia was interrupted. The results in the few cases in which this method of treatment has been used in the human, however, have been too unreliable to warrant its adoption.

The fourth operative procedure employed has been much more successful. Heyrovsky first proved the practicability of anastomosing the dilated esophagus and fundus of the stomach. His cases demonstrated that when this is undertaken below the diaphragm it is both a practical method of attack and is relatively safe. In the earlier attempts at making such an anastomosis, the stomach was carried through the diaphragm and the anastomosis was made intrathoracically. This was associated with a higher mortality rate than would probably result today. However, there seems to be no particular technical advantage to the supradiaphragmatic approach and the abdominal approach has had a low mortality rate. Continental surgeons have adopted esophagogastrostomy in numerous instances and, in general, with excellent results. In America, however, it has not received the attention that it deserves. in spite of the excellent case reports of Lambert[26] in 1914, and Watts[27] in

1923. In addition to these two cases, gastrojejunostomy for idiopathic dilatation of the esophagus has been reported by American surgeons in sixteen other instances, Keller[28] adding one case, Churchill[29] two, Womack[30] two, Ochsner and DeBakey[25] two, Gray and Skinner[31] one, and Janes[32], in the discussion of the last paper, added eight from the Toronto group operated

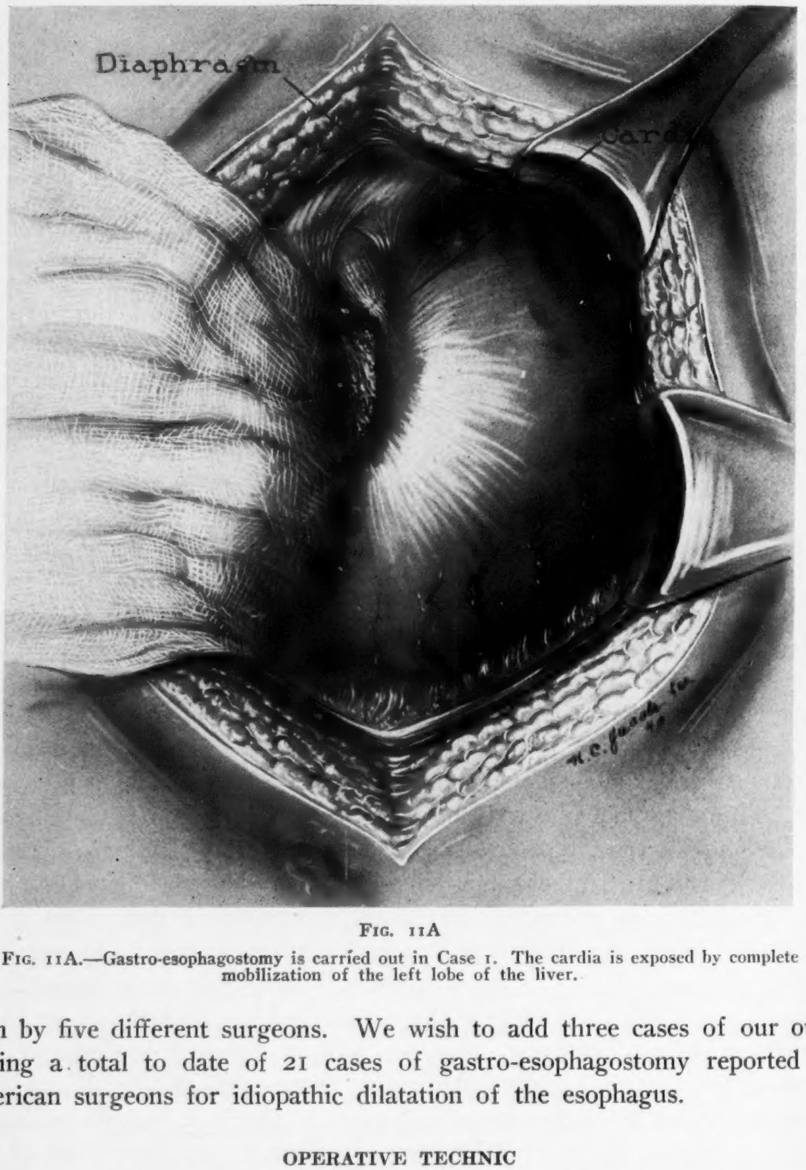


FIG. 11A

FIG. 11A.—Gastro-esophagostomy is carried out in Case 1. The cardia is exposed by complete mobilization of the left lobe of the liver.

upon by five different surgeons. We wish to add three cases of our own, making a total to date of 21 cases of gastro-esophagostomy reported by American surgeons for idiopathic dilatation of the esophagus.

OPERATIVE TECHNIC

The operation is a cardioplasty uniting the cardiac end of the stomach and the esophagus, similar to the Finney pyloroplasty (Fig. 11). The operative technic has already been well described in the American literature.[33–35]

The technic which we employed has been briefly published elsewhere.[35] We wish to call attention only to a few details: (1) The abdominal approach is superior for the ordinary case. (2) Adequate exposure is easily obtained by mobilization of the left lobe of the liver. In doing this, the left triangular ligament should be divided above the liver not only in its flat part but on each side as the two peritoneal surfaces start to separate in a "Y". This enables thorough mobilization of the left lobe of the liver. (3) A tape is passed around the cardia without injuring the veins. By traction on this

FIG. 11 (*Cont'd*)

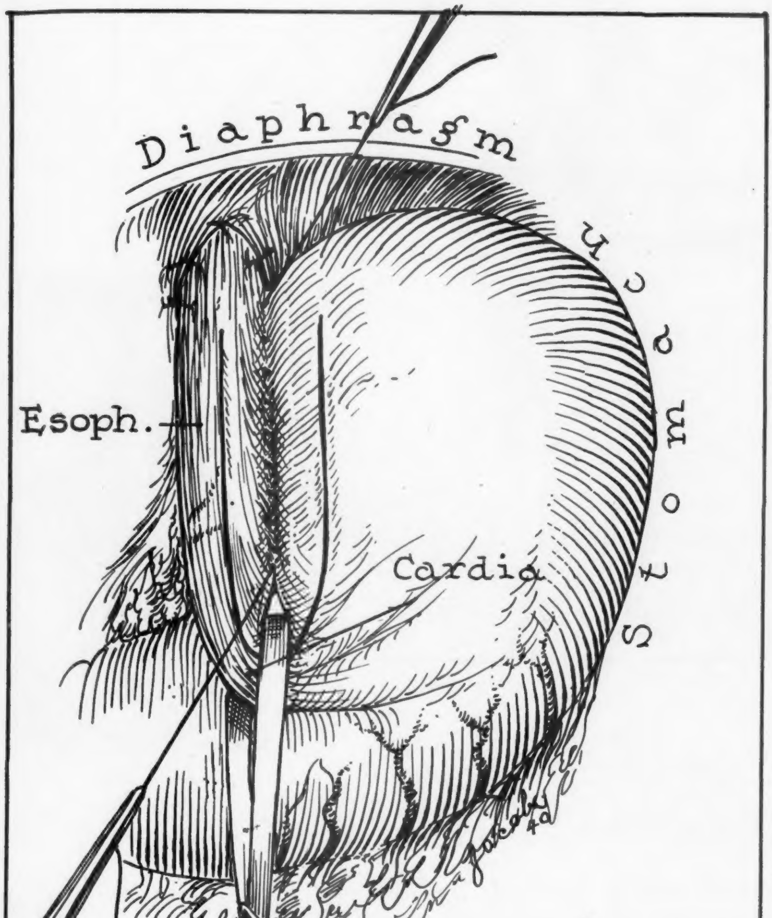


FIG. 11B

FIG. 11B.—After passing a tape about the cardia the peritoneum has been divided at the esophageal hiatus. The lower end of the esophagus is mobilized by blunt dissection and at least two inches of it pulled down into the abdomen. The crura of the diaphragm have been reattached to the esophagus at this higher level and the outer row of the posterior layer has been completed. The line of the incision is indicated by the "U."

tape, the peritoneal fold at the esophageal hiatus is easily made taut for division. After mobilization of the lower end of the thoracic esophagus by blunt dissection, two inches of the latter can easily be brought down into the abdomen. This traction is maintained throughout the first part of the

FIG. 11 (*Cont'd*)

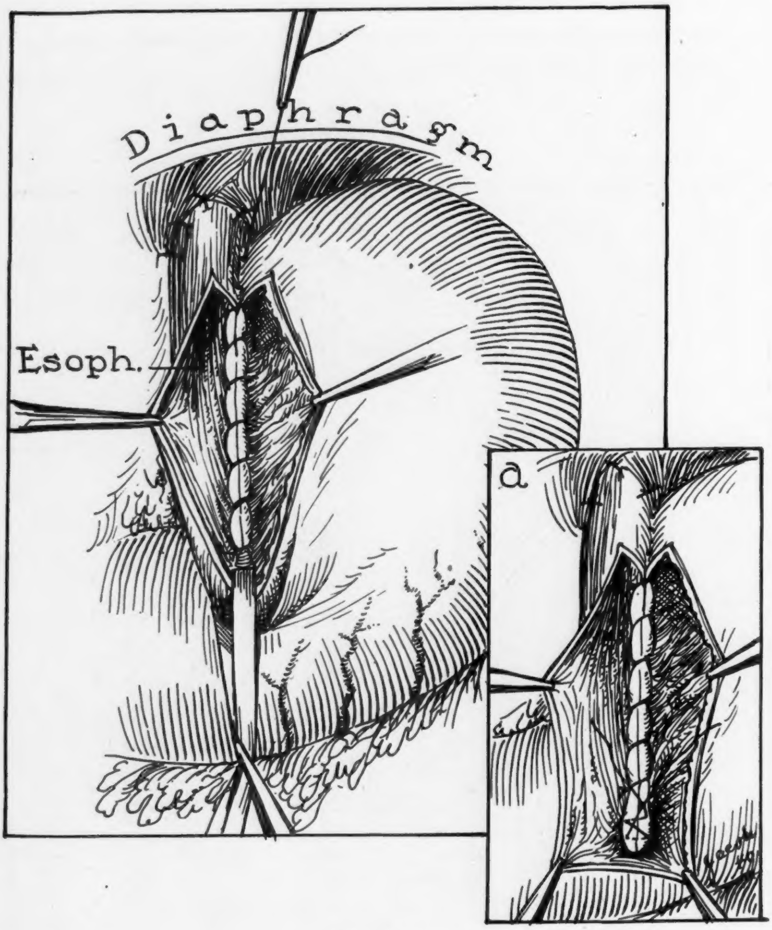


FIG. 11C

C. The stomach and esophagus have been opened except for the small bridge where the tape still maintains traction while the stomach and esophagus are sutured posteriorly. The tape and bridge at the cardia are now divided and one suture completes the outer posterior row.

anastomosis and in fact until the two rows of the posterior part of the anastomosis are completed except for the small bridge of tissue under the tape at the cardia. The tape is then removed, this bridge is divided and the posterior layer is completed with one figure-of-eight stitch. (4) It has not been necessary in our experience to tie off the esophagus with a tape, as advocated by Ochsner. The divided peritoneum is reattached to the esophagus at a higher level before the stomach and esophagus are opened and the parts

are well walled-off. The esophagus should be cleaned out carefully before operation but, even so, there may be some food residue in it which should be removed by suction. (5) After completion of the anastomosis it is not necessary to reattach the left lobe of the liver and in our cases drainage has not been necessary.

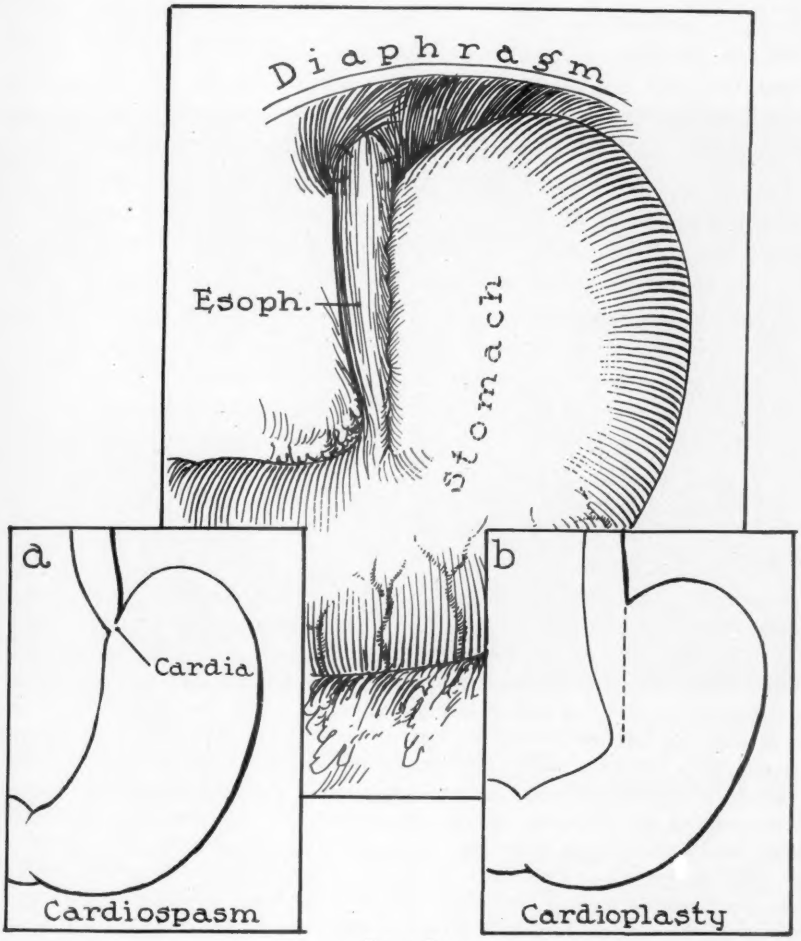


Fig. 11D
D. The two rows of the anterior segment have been completed making a wide opening between the esophagus and stomach.

RESULTS

Gastro-esophagostomy was carried out in this manner in three cases that failed to be relieved by repeated dilatations. In Case 4 a firm fibromuscular band was found at the pylorus. The pylorus itself when it was opened would not allow the passage of an ordinary lead pencil. Following operation the patient had a rapid convalescence. A Levine tube was left through the anastomosis for seven days. She was discharged from the hospital 19 days after operation eating a mixed diet. She rapidly gained 30 pounds in

weight, and is entirely relieved of her difficulty on eating, now six years after operation. Roentgenologic examination six months after operation showed that the dilatation of the esophagus had practically disappeared (Fig. 9). Repeated six years after operation, the cardia appeared normal. There was slight redundancy of the esophagus seen best in the supine position. In Case 5, the patient had a rapid convalescence. She left the hospital on the 23rd postoperative day, with marked improvement in her ability to eat but not complete cessation of symptoms, probably due to the fact that the operation had not affected the upper portion of the "S"-shaped curve. However, it has been found that the upper pocket can be emptied into the lower, and the patient gets relief from her symptoms if she lies on her side for 15 minutes after meals. She began eating a general mixed diet, which she was unable to do previously, and gradually gained weight. Six months after her operation she was having no trouble taking food if she did not eat too large an amount at a time. Now, six years after operation, she is quite a feeble old lady, being 84 years of age, but has had no major difficulty with her eating during this interval. It has not been possible to get a late follow-up of the roentgenographic appearance. Six months after operation the roentgenograms showed a definite improvement of the dilatation of the lower segment of the esophagus but little change in the upper pouch of the "S"-shaped curve, the latter would empty, however, on postural drainage. Barium entered the stomach much more quickly than before operation.

Case 6.—J. R., a 34-year-old French machine inspector, was admitted to the hospital, November 11, 1944, on account of difficulty in swallowing for the past three years. At first he was able to take solid foods if he washed them down with fluids. One and one-half months ago, he began having difficulty even getting liquids down. Diagnosis of cardiospasm was made on roentgenographic evidence in the Cleveland Clinic a year before admission. He had had seven dilatations during this year. At first he had improvement for several days following each dilatation, but for the last three or four times he had had very little improvement and the last time none at all. He has had vomiting spells every three weeks and frequently regurgitates his food. When he has the greatest difficulty in eating he develops a pain underneath the xyphoid accompanied by dyspnea and palpitation. He has lost 10 or 12 pounds during the present illness. Roentgenologic examination three months before admission showed a marked increase in the degree of dilatation and he was referred here as a candidate for operative treatment.

In the hospital it was found that the patient could not tolerate liquids at all well, regurgitating them every 10 to 15 minutes after ingestion. On physical examination the only finding of significance was the loss of weight. Roentgenologic examination on admission showed marked dilatation with some tortuosity of the lower half of the thoracic esophagus coming down to a smooth conical ending (Fig. 12). There were no definite peristaltic waves, though on deep inspiration, a variation in tone of the lower portion of the dilated esophagus could be seen.

Esophagoscopic Examination: The esophagus showed marked dilatation. The lower part was filled with solid and semisolid food remnants. The stomach was entered easily with the esophagoscope. The cardia appeared normal. There was no evidence of stricture or malignancy.

Esophagogastrostomy (Finney pyloroplasty type) was performed on November 15,

1944. The operative findings showed no constriction at the cardia and no evidence of any other pathology in the abdomen. He had a normal convalescence following gastro-esophagostomy. No indwelling tube was put in at the time of operation. He was taking fluid well on the fourth day after operation, and was discharged from the hospital on the 15th postoperative day. Six months after operation he was having no trouble whatever in eating. He had gained 24 pounds in weight. On roentgenologic examination barium passed freely into the stomach though dilatation of the esophagus was still present but much less in extent than before operation. The cardia of the esophagus appeared of normal width, without the sharp conical shape noted before operation (Fig. 13).

FIG. 12

FIG. 13

FIG. 12.—Case 6: Dilatation of the esophagus before operation.

FIG. 13.—Case 6: Roentgenogram of the esophagus and cardia six months after operation shows disappearance of the sharp conical end of the esophagus. Dilatation of the esophagus is less but is still present. Cardia is of good width.

Thus, three cases of idiopathic dilatation of the esophagus, entirely resistant to the usual conservative treatment including repeated dilatations, have been relieved of all or the major part of their dysphagia by gastro-esophagostomy. Case 4 was an example of the group with a band constricting the cardia. Case 5 was an example of the dolicho-esophagus group with an "S"-shaped curve and pooling at two levels. Case 6 apparently was an example of the achalasia group which did not respond as most of this group do to esophageal dilatation. Operative relief of these patients is not unduly difficult nor too dangerous. The latter fact is attested to, I believe, by the successful result in Case 5 at the age of 78.

It is interesting that the relief of symptoms does not require the disappearance of the dilatation of the esophagus. In fact, the prompt return of the esophagus to a normal width, as occurred in Case 4, is somewhat

unusual. The other cases (Nos. 5 and 6) had a marked improvement in the emptying of the dilated esophagus into the stomach and some decrease in the dilatation. This is the usual experience as reported in the literature.

CONCLUSIONS

Dilatation of the esophagus without any clinically discoverable obstruction is not a disease entity but is a symptom-complex. At least four clinical types can be recognized, each with a different etiology: namely, (1) achalasia of the esophagus; (2) true cardiospasm; (3) partial constriction near the cardia; and (4) dolicho-esophagus.

Achalasia is probably the most common type. It usually responds well to dilatation. In true cardiospasm the reflex originating focus should be discovered and removed. Those cases caused by a partial constricting band near the cardia are few in number but are probably resistant to conservative treatment. Dolicho-esophagus, an "S"-shaped lengthening with pooling of esophageal contents at different levels, should probably be operated upon immediately on recognition. This type is very difficult to control when the dilatation becomes gigantic and is complicated by infection of the esophageal wall.

Subdiaphragmatic esophagogastrostomy (of the Finney pyloroplasty type) appears to be the operation of choice in those cases of idiopathic dilatation of the esophagus that are resistant to the usual conservative measures. The symptomatic results of this operation are usually excellent whether or not the dilatation of the esophagus is entirely corrected. The danger of the operation is not excessive when proper precautions are taken. After this operation has been employed more extensively, the number of cases where it is indicated will probably be considerably increased but it should never replace conservative dilatation in the majority of cases.

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CONGENITAL OR HEREDITARY POLYPOSIS OF THE COLON*

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In recent years our interest in the subject of multiple polyposis of the colon has been greatly aroused due to the fact that five patients with this disease have come under our care. There was, unquestionably, an hereditary tendency in four of these cases, and no history of an hereditary background in the fifth. We are particularly interested in the congenital or hereditary type of this disease, and the greater portion of this paper will be devoted to that phase of the condition. An hereditary tendency has been reported in 50 per cent of the cases of multiple polyposis of the colon, but we believe that if sufficient and accurate information could be secured from the family in question, the hereditary tendency would be much greater. This does not apply to cases of solitary polypi or a few scattered polypi in the colon or rectum.

Diffuse polyposis of the colon has been recognized as a disease entity for many years. Menzel, according to Warwick,[2] first called attention to this fact in 1721. Lushka,[3] in 1861, reported a case in which there were thousands of polypi in the colon of a woman, age 30. As Mayo and Wakefield[4] state, this is the most convincing report of the disease by an early writer. Harrison Cripps,[5] in 1882, was the first to record that polyposis of the colon occurred in several members of one family.

CLASSIFICATION

The classification of this disease leaves much to be desired. However, the two classifications that are most widely accepted are those of Erdmann and Morris,[6] and Wesson and Bargen.[7] Erdmann and Morris divide polypi of the colon into two groups: (1) Those which are adolescent, or congenital; and (2) those which are adult, or acquired. Wesson and Bargen divide polypi into (1) true polypi; and (2) postinflammatory polypi. Fitzgibbon and Rankin[8] classify polypi on the basis of their gross characteristics and histologic nature. These classifications make it emphatic that there are two types of polypi in the colon.

ETIOLOGY

The congenital or hereditary type of multiple polyposis of the colon is a definite clinical entity. The disease may be present in both males and females, more commonly in the male, and it may be transmitted by both sexes. Instances of multiple polyposis in more than one member of a family have been reported by numerous writers.[4, 6, 9, 10, 11, 12, 13, 14, 15, 16, 17] No patient has been known to be born with this disease. McKenney[10] reported a case of a boy, age 2, who had this disease, this being the youngest case on record. Evi-

* This paper was to have been presented before the Annual Meeting of the American Surgical Association, May, 1945.

dently, individuals are not born with this disease, but develop it later in life. None of our cases developed symptoms before the age of 14. Lockhart-Mummary[9] stated that he sigmoidoscoped a man, age 39, because he came from a family with this disease. The colon was normal, but four years later marked polyposis was present. The disease does not have to appear in early infancy or childhood to prove the question of heredity. Certain dominant characteristics do not manifest themselves until after puberty, or even middle age.

The most logical explanation of the hereditary aspect of this disease is that there occurs a mutation of the genes in an individual who has not exhibited the disease, and he passes the mutated genes on to his children. The genes may be passed as a dominant or recessive characteristic. The disease will occur in every generation if the genes is dominant; if it is recessive, both parents must carry the mutated genes before the condition will be seen. Lockhart-Mummary,[9] who expounded this theory, surmised that in multiple polyposis the genes is a mendelian dominant. Bernstein's[16] cases show that the disease may appear in one generation, skip the second, only to reappear in the third.

When the disease is found in an individual, all members of the family should be examined thoroughly for the presence of polypi. Several cases have been reported in recent years when the initial history failed to show the presence of the disease in other members of the family, but subsequent questioning revealed the disease in other close relatives.

Most writers agree that acquired multiple polyposis of the colon is secondary to irritation or infection, and that chronic ulcerative colitis is the disease which most commonly causes polypi of the colon to secondarily develop. Rankin[18] reported diffuse polyposis of the colon in 69 of 693 cases of chronic ulcerative colitis. Bargen and Coffey[15] reported a series of 417 patients with chronic ulcerative colitis. Fifteen point eight per cent of this group developed polypi of the colon, and 16.6 per cent of this group had disseminated polyposis.

PATHOLOGY

In polyposis, the polypi vary in size from 1 mm. to 1–2 cm. in diameter. They may be pedunculated or sessile, the pedunculated polypus having a pedicle or stalk. In congenital multiple polyposis, the polypi are distributed throughout the colon. The specimen removed from one of our patients is shown in Figure 1. In our cases, the sessile type has predominated. All of the polypi are first sessile in nature, and many of these become pedunculated. The acquired polypi are more irregular in size and shape, more scattered, and fewer in number than the congenital polypi.

RELATION OF THE POLYPI TO MALIGNANCY

Friedell and Wakefield[1] state that all polypi of the colon probably will become malignant if the patient who has them lives long enough. Many

writers agree with this statement. Bargen and Coffey[15] found that carcinoma is much more likely to develop in congenital multiple polyposis than in the acquired form, the relationship being 82.8 per cent to 21.9 per cent in their two series of cases.

Proof of the tendency of the polypi to become malignant is shown by the reports of many writers. In a series of 19 cases of congenital multiple polyposis reported by Mayo and Wakefield,[4] six developed carcinoma. McKenney[10] reported carcinomatous change in 33.3 per cent of his cases. In 1928, Hullsiek[14] reported that of 128 cases of multiple polyposis of the colon in the literature, 46 developed carcinoma. Numerous other writers have reported cases of polyposis with malignant changes.[6, 11, 12, 13, 16, 19, 20, 21, 22]

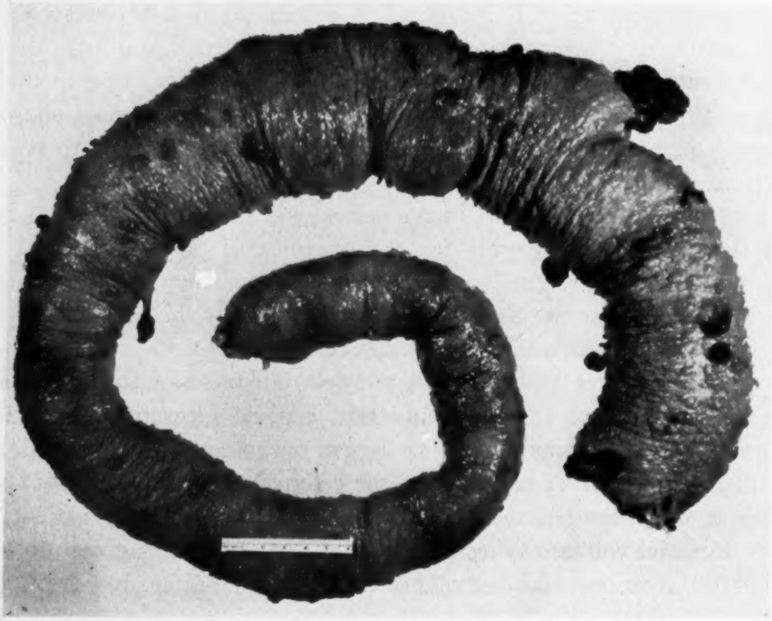


FIG. 1.—Specimen of large intestine removed from Patient No. 1 at operation. The mucosa is diffusely covered with countless wart-like nodules which vary in size from pin point to 1 × 2.5 cm., many of them confluent. Their elevation above the plane of the mucosa varies from a fraction of 1 mm. to 3 or 4 mm.

They are greyish-red in color and fairly firm in consistency. The mucosa between the wart-like elevations is not thickened, but pale, smooth, and glistening. There are also numercus sessile and pendunculated polypi, representing apparently increase in size of the wart-like growths.

The polypi project above the plane of the mucosa from 5 to 4 cm. and their cross section area varies from 2 × 4 mm. to 2.6 × 3.2 cm. The large polypi are dendritic in character, dusky purplish-red in color, and firm in consistency.

Numerous sections were taken from the growth with an idea of showing the evolution of the polypi.

Cattell[23] quoted Ewing as saying that "nowhere else can the change from normal mucosa to inflammation, gland cell hypertrophy, adenoma, and adenocarcinoma, be so clearly demonstrated as in multiple polyposis of the colon." These changes are well illustrated in Figures 2–6 which are sections of polypi removed from one of our patients. (E.H.)

We have found that the larger polypi have a greater tendency toward malignancy. Malignant change is suggested by ulceration, nodular irregularities, and a broad pedicle and base. Grossly, firmness, induration, and fixation are suggestive of malignancy.

In addition to the polyposis, two of our cases showed large carcinomatous masses present in the colon, one of which was producing partial obstruction.

SYMPTOMS

The early symptoms in this disease are very mild, and, therefore, the patient is not likely to present himself for examination and treatment until the symptoms have been present for many years. Two of our patients had symptoms for seven years before consulting a physician. Because of the mildness of the early symptoms, some of the polypi may have undergone malignant changes before medical consultation is sought, as mentioned above. One of our cases had a carcinoma of the stomach and another an obstructing lesion of the rectosigmoid before seeing a physician. Both had practically no symptoms due to the colonic polypi.

As Slaughter[24] stated, polypi can produce any of the reactions of colonic or rectal irritation. Rectal bleeding and diarrhea are probably the two most common complaints. The bleeding may be mild or severe in character. One of our patients had a massive hemorrhage before consulting a physician. Bargen and Coffey[15] obtained a history of rectal bleeding in 62 per cent of 29 cases. Frequent loose stools may be the only symptom in some cases. Four of our five cases complained of mucus in the stools. Intermittent attacks of crampy, abdominal pain are common. Other more vague complaints that may be present are nausea, indigestion, easy fatigue, loss of weight, and loss of appetite.

A summary of the complaints in our five patients is shown in Table I.

TABLE I

SYMPTOMS IN FIVE CASES OF MULTIPLE POLYPOSIS

Complaint	No. of Cases
Abdominal cramps	4
Mucus in stools	4
Blood in stools	3
Diarrhea	3
Weight loss	3
Nausea	2
Loss of appetite	2
Indigestion	2
Easy fatigue	2
Vomiting	2

DIAGNOSIS

Complete examination of the rectum and colon is essential for the diagnosis of this condition. When one presents complaints suggestive of a large bowel lesion, the physician may be suspicious of a condition of multiple polyposis,

FIGURES 2—6 ARE SECTIONS OF POLYPI TAKEN FROM PATIENT 1. THEY ILLUSTRATE STAGES IN THE DEVELOPMENT OF INTESTINAL POLYPI. (LOW POWER AND HIGHER MAGNIFICATION ARE SHOWN.)

A

B

C

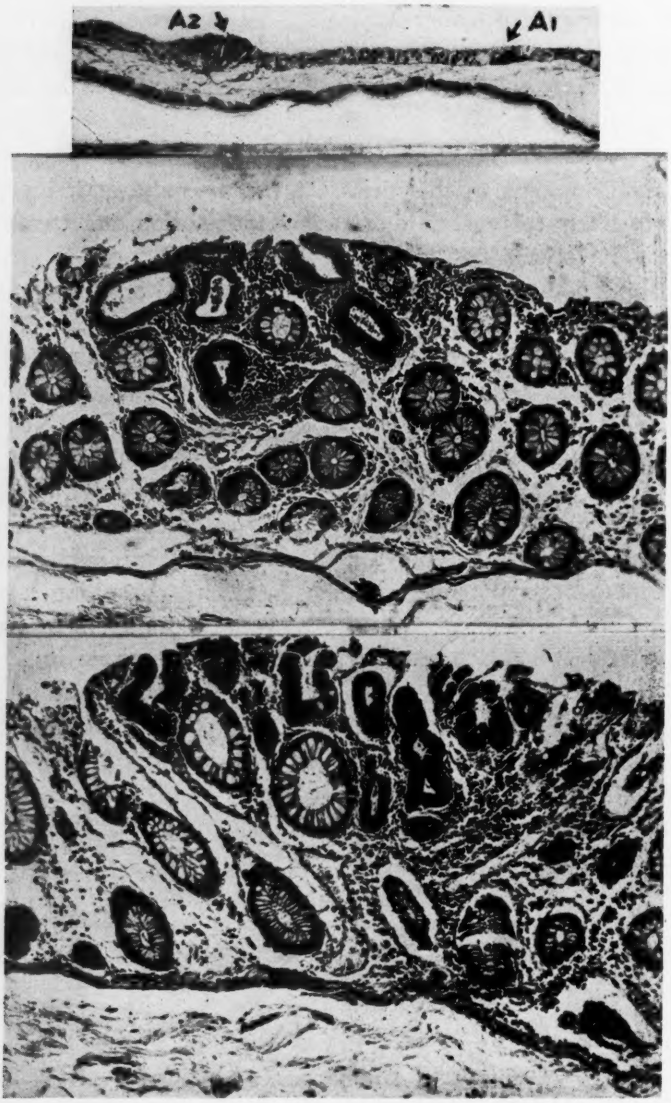


FIG. 2.—A is a low power view of a section of the wall of large intestine showing beginning of polypi at two places A1 and A2. A1 is a barely visible, slightly darker area, surrounded by normal mucosa. A2 is a larger more elevated area.

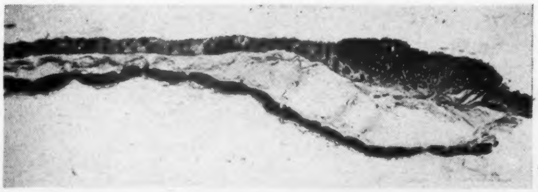
B is a magnification of A1 and shows very slight thickening of the mucosa due to fairly localized infiltration of small round cells. The cells of these glands do not contain or secrete any mucus and stain rather deeply. The nuclei are more prominent, slightly hyperchromatic, with a suggestion of loss of polarity.

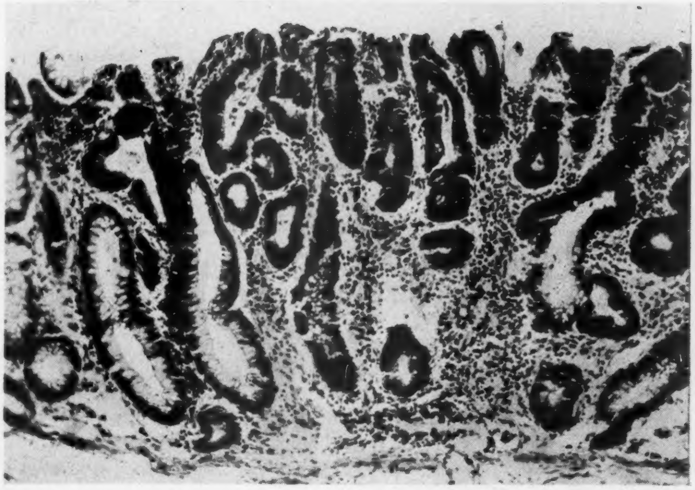
C is a magnification of A2. The changes are slightly further advanced. Round cell infiltration is present and abnormal glands are more numerous.

but since there are no symptoms pathognomonic of polypi of the colon, the above complete studies must be done to make this diagnosis.

Any patient with complaints referable to the lower bowel should have a digital, proctoscopic and sigmoidoscopic examination as a minimum. In multiple polyposis of the colon, polypi can be felt digitally and seen through the proctoscope and sigmoidoscope. There is no difficulty in detecting polypi

A





B

Fig. 3.—A is a low power view of a definite polypus formation with the beginnings of a connective tissue core.
B. High power magnification of A. This shows the junction of polypus with fairly normal mucosa.

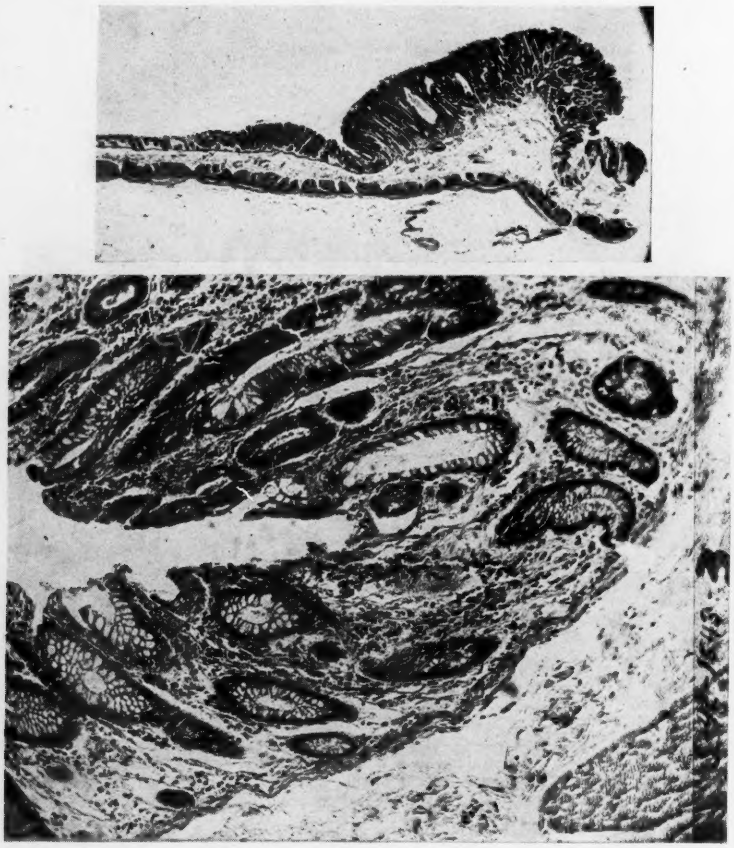
through the sigmoidoscope. They may be pedunculated in nature or simply elevations of the mucous membrane. Often they appear in clumps and are variable in size.

If polypi are felt on digital examination or seen through the sigmoidoscope, roentgenologic examination of the colon is indicated. Before a barium enema is given, the colon must be absolutely clean, since polypi can be hidden from view by fecal particles. We give routinely an ounce of castor oil the night before the examination and an enema the morning of the examination. To adequately and completely demonstrate polypi of the colon, the barium enema should be followed by postevacuation films and then by air insufflation

and double contrast films. Figure 7 shows roentgenologic studies performed on one of our patients; the value of double contrast films is well illustrated.

It is important to realize that an individual with multiple polyposis of the colon may also have a lesion in the stomach or small intestine. One of our cases had a carcinoma of the stomach, and several cases have been reported in the literature of an isolated polypus in some portion of the small intestine.

A



B

Fig. 4.—A is a low power view of a flat, fairly sharply defined elevation surrounded by normal mucosa.

B. Magnification of A. The changes are essentially the same as in Figure 3B. There are a few hyperemic capillaries.

For this reason, we recommend roentgenologic examination of the stomach and small intestine in an individual with multiple polyposis.

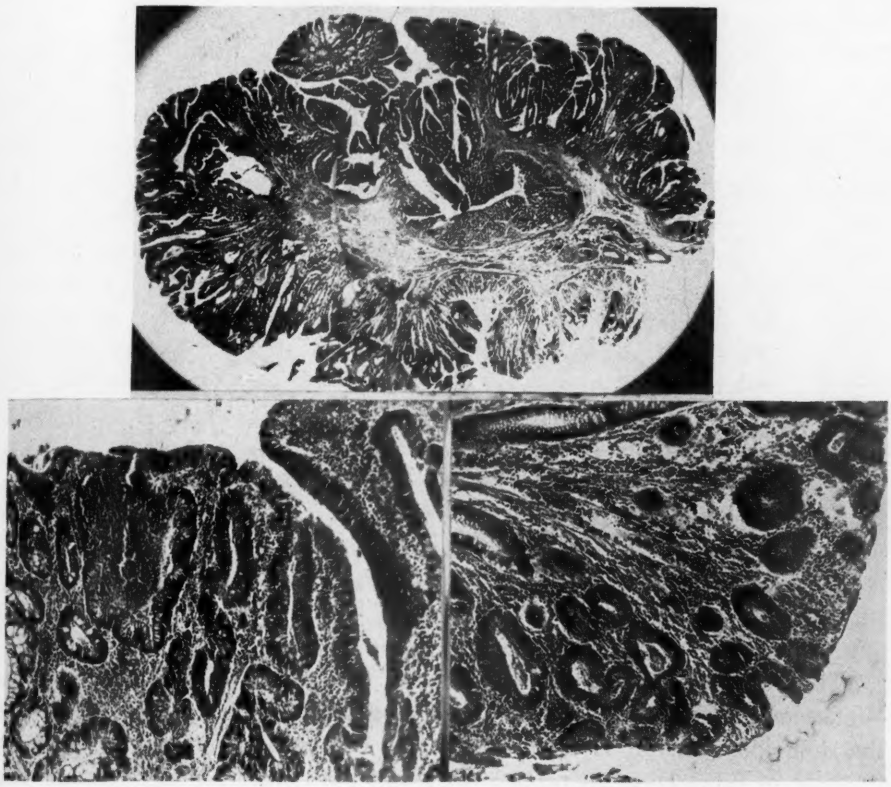
The possibility of a malignant change is often suggested by roentgenologic examination. Likewise, the evidence of malignancy, if present, may be obtained from a biopsy of a polypus. A biopsy should always be taken of a suspicious appearing polypus.

TREATMENT

The treatment of congenital polyposis of the colon is a surgical problem. Surgical removal of the involved colon offers the only hope of permanent relief to those individuals with this disease. This statement is made because it is a well-known fact that polypi of the colon tend to undergo malignant changes. The affected portion of the bowel must be removed.

The surgical treatment of this disease has undergone many changes and advances. Ileostomy, which was the first form of treatment, evidently was not the answer. It merely prolonged the agony, as it was usually done late

A



B C

FIG. 5.—A shows a large dendritic polypus with a well-developed connective tissue core. B & C are magnifications of A showing essentially the same picture described above on a larger scale.

in the disease. It was soon realized that in order to obtain anything of a permanent value, it was necessary to remove the affected portion of the bowel. Ileostomy, followed by total colectomy, then became the procedure of choice. This was not totally satisfactory because many individuals could not satisfactorily tolerate a permanent ileostomy.

Our mode of treatment of these cases is not original, but we believe that it is the most logical and satisfactory method. Our plan is as follows: The polypi in the rectum and rectosigmoid area are destroyed by fulguration. Following this an ileorectosigmoidostomy is performed; and finally a colectomy down to the anastomotic site is done. In this plan the normal outlet of the rectum and its sphincters is preserved, and as Mayo and Wakefield[4] state, the rectosigmoid and sigmoid flexure, which contain the nervous mechanism which controls the desire to defecate, are left intact. A schema of the plan of treatment is shown in Figure 8.

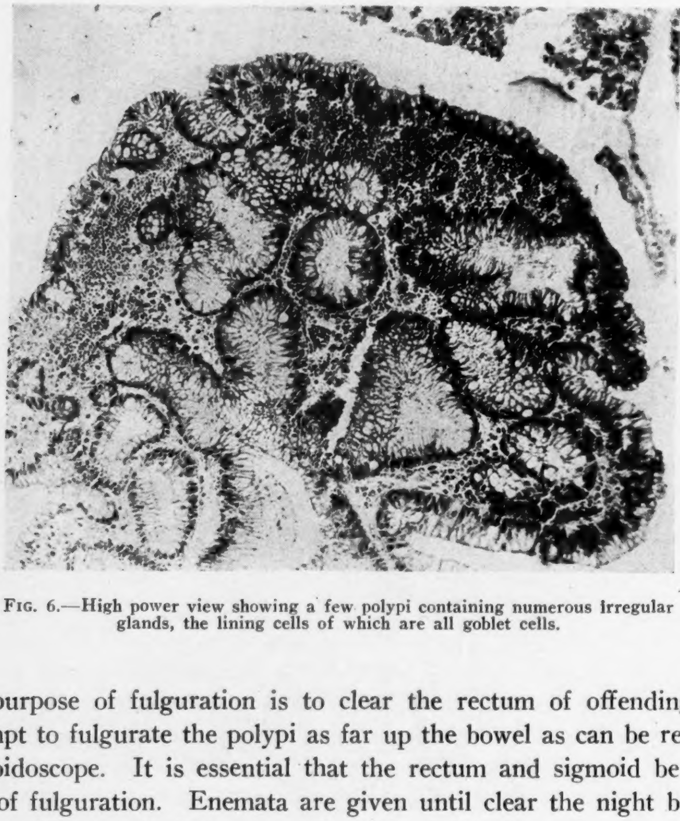


FIG. 6.—High power view showing a few polypi containing numerous irregular glands, the lining cells of which are all goblet cells.

The purpose of fulguration is to clear the rectum of offending polypi. We attempt to fulgurate the polypi as far up the bowel as can be reached by the sigmoidoscope. It is essential that the rectum and sigmoid be clean at the time of fulguration. Enemata are given until clear the night before the procedure. No anesthetic is given since the full coöperation of the patient is necessary. The patient is placed in the prone, inverted position. We believe that the patient should be hospitalized because hemorrhage and perforation may occur when diathermy is applied to this portion of the bowel. These two complications usually occur 5–10 days after fulguration. It is not possible to say how many polypi may be fulgurated at a time or how frequently fulgurations should be performed. These depend on many factors. Some patients can tolerate the procedure better than others. Likewise, the healing process in the mucous membrane of the rectum varies in different individuals.

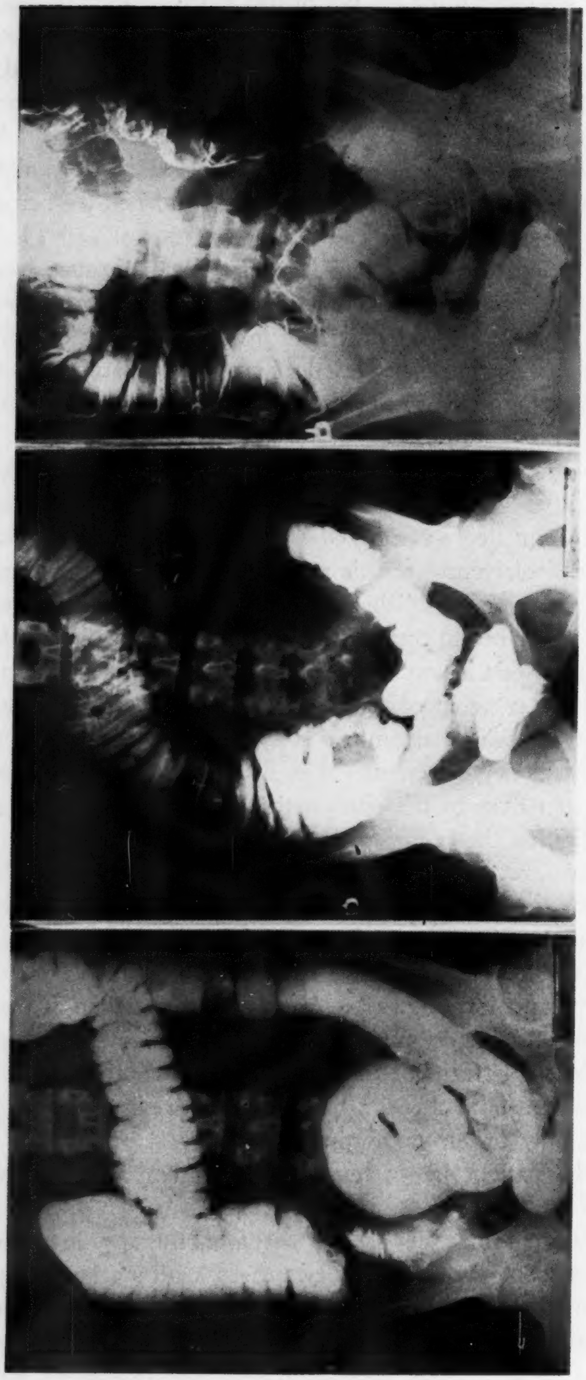
We have found that if fulgurations are performed more frequently than every three days, the mucous membrane will still be inflamed and edematous, and the individual will suffer discomfort. As a rule, however, individuals withstand fulguration of the polypi quite well. We usually fulgurate for 30 to 45 minutes unless the patient complains of fatigue before that time. Following fulguration, the patient may develop a mild fever and low abdominal pain, but these usually quickly disappear. We have had neither hemorrhage nor perforation as a complication.

After we are satisfied that the polypi for a sufficient distance have been removed by the method of fulguration, we allow the patient to go home for at least a month. This is done to allow the inflammation incidental to the fulguration to subside. During this period at home, the patient is urged to follow a high-vitamin, high-caloric diet, and to get plenty of rest and sunshine. Supplementary vitamins are prescribed.

When the patient returns to the hospital, he is again sigmoidoscoped. If the lower segment of the bowel appears satisfactory and the general condition of the patient is good, an ileorectosigmoidostomy is performed. It has been proposed to do a hemicolectomy[4] at this stage, but since the most hazardous part of this procedure is to establish an anastomosis of the ileum with the terminal bowel, we prefer not to complicate this stage with an hemicolectomy. Continuous spinal anesthesia is employed. The terminal ileum is anastomosed to the upper end of the segment of sigmoid that had been freed of polypi by fulguration. An end-to-side anastomosis is the one of choice. A six-inch segment of the terminal ileum is removed in order to more easily swing the distal end of the ileum over to the site on the rectosigmoid. Two to three weeks later, depending on the postoperative course of the patient, the remaining colon is removed down to just above the anastomosis between the ileum and rectosigmoid, the latter being closed. It is important to know that after fulguration polypi may appear from time to time, and these have to be removed by fulguration. Lewis[11] reported a case that is illustrative of this point.

Buie[25] recommends performing the colectomy first, and then, if the patient survives, the rectum and rectosigmoid may be prepared by fulguration and later ileosigmoidostomy performed. He advocates this particularly because in some cases the patient goes through endless time, discomfort and preparation by fulguration only to succumb when colectomy is performed. If this plan is followed, the patient has a temporary ileostomy, and often this is difficult to handle.

Some men have reported the use of roentgenotherapy in these cases. VanZant[26] employed roentgenotherapy in two cases, with marked symptomatic relief and reduction in the number and size of the polypi. McKenney[10] reported similar results in ten cases treated by roentgen ray. According to McLaughlin,[13] no five-year cures have been reported that were treated by roentgen ray, and severe systemic reactions have occurred. The basis for the employment of roentgenotherapy is that hyperplasia of the lymphatic



A B C

FIG. 7.—X-ray films of Patient No. 3

A. Barium enema study. A large polypus is visible in the descending colon.

B. Postevacuation film.

C. Film taken after air-insufflation.

tissue and lymphatic infiltrations are present, and these are both susceptible to the roentgen ray.

The following five cases are reported, four of which have a definite hereditary background.

CASE REPORTS

Case 1.—E. H., a 29-year-old male, from Mississippi, was admitted to the Service of Dr. Damon B. Pfeiffer, at the Abington Memorial Hospital, August 5, 1942, with a chief and only complaint of diarrhea. He stated that this diarrhea began eight years ago and was characterized by three to four movements per day, the stools being loose, containing mucus but no blood. Five years ago he began to have severe abdominal cramps. Roentgenologic examination of the stomach at that time was negative. A proctoscopic examination revealed "growths in his rectum," as he expressed it. Shortly after this, in 1937, he decided to go to a large clinic for a check-up.

After a complete gastro-intestinal study had been done, he was told that he had multiple polypi of his colon and rectum. It was decided to fulgurate these polypi from his rectum and sigmoid, and after this was completed to do a colectomy. For two or three times a week for two months, fulguration of these polypi was performed. Because of financial difficulties, the patient left and returned home. His diarrhea had decreased in intensity to two, or less, movements a day, the stools being more formed, containing mucus but no blood. The abdominal cramps disappeared, and his weight was maintained.

Following this, his weight increased 45 pounds in one year. For the next four years his physical condition was excellent. In 1940, a gastro-intestinal series was repeated, and his local physician advised against operation. A year later the diarrhea returned, the stools numbering three to six per day. He began to suffer from abdominal cramps again, and a gradual weight loss occurred. In addition, he began to have rectal bleeding. The intestinal roentgenologic examination was repeated, and some of the polypi appeared to have increased in size, and operation was advised.

Further questioning revealed that the patient was an only child. His mother had died of "cancer of the bowel," 13 years ago, as did his maternal grandmother. The patient thinks that his maternal aunt has a similar condition. We have no proof of these facts, other than what the patient has told us. His father was given a clean bill of health. The hereditary background is shown in Figure 9.

Physical Examination: General examination at the time of admission to our hospital revealed a 29-year-old white male, 5 feet 10 inches tall, weighing 125 pounds. He appeared in excellent health. Examination of the heart and lungs was negative, as was the abdominal examination. Digital examination of the rectum, likewise, was negative. Proctoscopic and sigmoidoscopic examinations revealed this portion of the large intestine to be negative, except for one polypus just above the rectosigmoid area. The blood count and other laboratory studies were within normal limits.

Course in Hospital; On August 7, 1942, (two days after admission), an ileorecto-sigmoidostomy was performed upon the patient by Dr. Damon B. Pfeiffer. Pre-operatively, the patient was given succinylsulfathiazole. His postoperative course was uneventful. Wangensteen drainage was maintained for three days postoperatively, and parenteral fluids were given for six days postoperatively.

On August 26, 1942 (19 days after the first operation), a second operation was performed—the procedure anticipated being a total colectomy. The anastomotic site was found to have been pulled over toward, and attached to, the terminal ileum and cecum by adhesions. A pocket of purulent material was found at this site, so it was decided that it would be unwise to try to free the adhesions. Therefore, the colon was resected from just proximal to the anastomotic site on the sigmoid, to within three or four inches of the blind stump of the ileum, leaving the terminal ileum and cecum *in situ.*

The cecum was brought out at the midportion of the incision. On the operating table, the patient received 750 cc. of blood plasma, and, later, 500 cc. of blood. For two weeks postoperatively he ran a slight elevation of temperature, but otherwise his condition was excellent. He had one to two normal bowel movements per day. His incision healed well, and he was discharged on September 23, 1942 (seven weeks after admission),

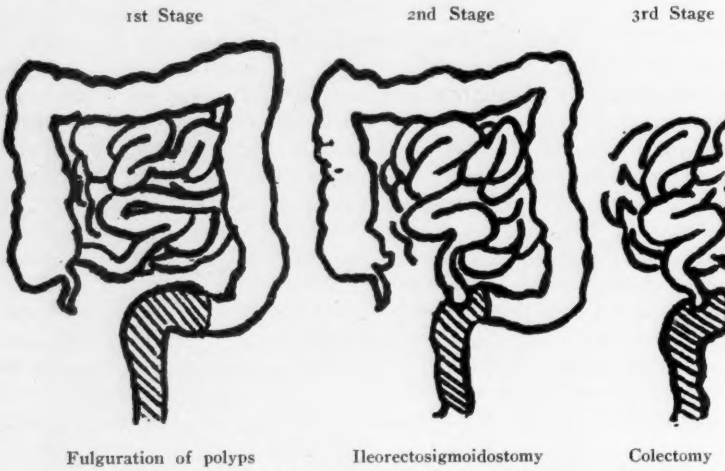


FIG. 8.—Schema of surgical treatment of multiple polyposis of the colon.

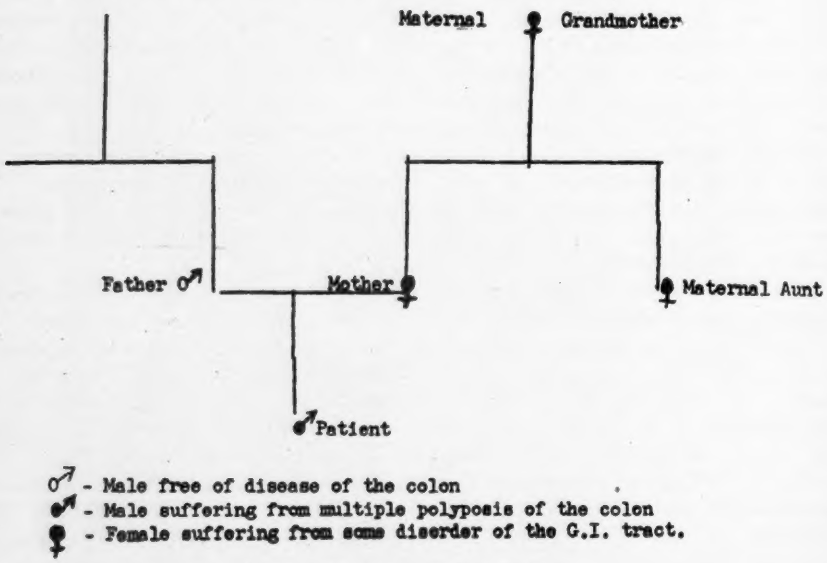


FIG. 9.—Family tree of Patient No. 1.

stating that he had better control of his intestinal tract than ever before. A digital examination of the cecum, just before discharge, revealed several small polypi to be present in this loop of bowel. These were removed by fulguration. The patient returned in July, 1944, and the cecum was removed under spinal anesthesia.

COMMENT.—The treatment in this case followed the plan outlined above. Following operation, the patient gained weight, had normal bowel movements

and offered no complaints. It is extraordinary that with so small a portion of the bowel remaining to serve as a receptacle for the fluid ileal contents, no diarrhea or irritation has resulted, and the patient reports that he has only one or two evacuations per day.

Case 2.—J. H., a white male, age 29, was admitted on the Surgical Service of Dr. Damon B. Pfeiffer, at the Abington Memorial Hospital, March 16, 1943, with a chief complaint of blood stools. Five months previous to admission, he began to complain of an "upset stomach." Foods did not agree with him, his appetite became poor, and he was occasionally nauseated. Since that time he had infrequently noted mucus in his stools and diarrhea. For three weeks before admission he had intermittent attacks of gas pains. During this three-week period he had noted blood in his stools daily and had one severe hemorrhage by rectum. No vomiting had occurred. A weight loss of ten pounds had occurred during the three weeks previous to admission. The remainder of his history was negative, except that his mother had died of "cancer of the intestines." On subsequent questioning of the patient's father, it was found that the mother had an intestinal malignancy, plus multiple polyposis of the colon. This hereditary background is shown in Figure 10.

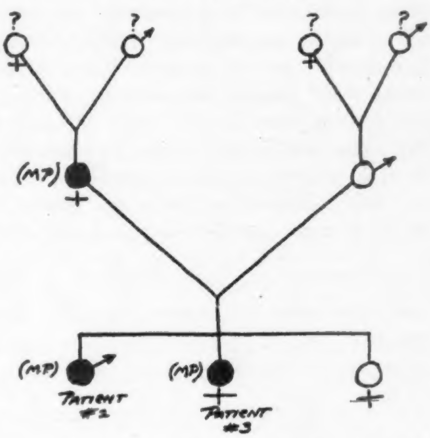


FIG. 10.—Family tree of Patients 2 and 3.

Physical Examination: This revealed a thin, pale, 29-year-old male, in no evident distress. Temperature, pulse and blood pressure were normal. The physical examination was entirely negative, except for many small polypi in the rectum, which were felt on digital examination.

Laboratory Data: Hemoglobin 88 per cent; white blood cells 7,400, with 58 per cent polymorphonuclears, 36 per cent lymphocytes, 1 per cent monocytes and 5 per cent eosinophils. Blood urea nitrogen 13; fasting venous sugar 80. Wassermann and Kahn negative. Urine negative, except for an occasional white blood cell per H.P.F.

Course in Hospital: A barium enema had been given to the patient at another hospital, and multiple polyposis of the colon was found to be present. Two days after admission, the patient was proctoscoped, and numerous pedunculated and sessile polypi were observed. Fulgurations were then performed as frequently as possible, although the patient was not very coöperative. Occasionally he would leave the hospital for several months.

On August 15, 1943, a mass became palpable in the left lower quadrant of the abdomen just above the outer half of the iliac crest. The patient's temperature remained normal, and the question arose whether this mass was an abscess secondary to fulguration or whether it was a malignancy of the sigmoid. A barium enema was done, and in addition to multiple polyposis throughout the colon, there was an irregularity in the contour of the sigmoid just above the level of the inferior margin of the sacro-iliac joint. This covered an area three centimeters long and 15 millimeters wide. The fulgurations were continued every two days, and the mass diminished greatly in size.

On October 15, 1943, the patient went home and did not return to the hospital until May 4, 1944, at which time four more fulgurations were performed, and the lower sigmoid, rectum and anus for a distance of seven to eight inches was now free of polypi.

On May 22, 1944, an exploratory celiotomy was performed under continuous spinal anesthesia, supplemented by intravenous sodium pentothal. A firm, indurated lesion was found at the junction of the rectum and sigmoid and was unquestionably malignant. The lesion was bound down to the posterior parietal peritoneum and had infiltrated the bladder. The intestine distal to the lesion was free of polypi on palpation, but the large intestine proximal to the lesion contained innumerable polypi. The liver was free of metastasis. The findings were otherwise negative. To liberate the malignant area, the posterior half of the bladder had to be resected. The left ureter was found to extend into the malignant tissue, and it was therefore clamped, cut and ligated proximal to the lesion. The large intestine was severed between clamps distal to the lesion with the cautery, and the stump of the distal segment closed in the Parker-Kerr fashion. This lower segment of bowel was pushed down into the pelvis. The descending colon was then severed proximal to the lesion, and the lesion was thus resected. A single-barrel colostomy was performed. A drain was placed in the pelvis, and the abdomen was closed in layers.

The postoperative course was stormy, and on the 12th postoperative day the patient died. Autopsy revealed extensive peritonitis.

COMMENT.—This patient's family history was investigated thoroughly, and is shown in Figure 10. His mother evidently suffered from the same disease entity. Later his sister was found to also have multiple polyposis of the colon. This patient was a problem in that he would leave the hospital and not return until several months later. We had contemplated doing an ileorectosigmoidostomy after the fulguration of the polypi and then a colectomy, but the findings at operation nullified these plans.

Case 3.—H. S., white, female, age 21, sister of J. H. (Case 2), was admitted to the Abington Memorial Hospital November 15, 1944. For five to seven years she had been suffering from diarrhea, and had noticed that her stools were never formed. In addition, she had noticed blood in her stools during this same period of time. For several years she had experienced hunger pains. During the six months preceding admission she had lost ten pounds in weight, although her appetite remained good. For several months, mucus had been present in the stools. Nausea occasionally occurred, but no vomiting. On June 4, 1944, her brother (Case 2) had succumbed following an abdominal operation for multiple polyposis of the colon, and this fact prompted her to go to a physician. Her hereditary background with relation to congenital polyposis is shown in Figure 10. A barium enema showed multiple polyposis of the colon (Fig. 7). At the time she was pregnant. A therapeutic abortion was deemed advisable, and this was performed on November 16, 1944.

The patient experienced the usual childhood diseases. At the age of three she had pneumonia, and two years ago she suffered from secondary thrombocytopenic purpura. She had undergone no operations.

Her mother died of "stomach trouble," which on investigation was found to be multiple polyposis of the colon. Her brother died following an operation for multiple polyposis of the colon and carcinoma of the rectosigmoid area.

Physical Examination: Patient was a pleasant appearing 21-year-old white female, who was two months pregnant. Temperature, pulse, respirations and blood pressure were normal. She was fragile in appearance, but in no acute distress. Her physical examination was negative, except for slight enlargement of the uterus, and the presence of multiple polypi of the rectum, as revealed by digital and proctoscopic examinations.

Laboratory Data: Hb. 11.4 Gm., 75 per cent; R.B.C. 3,630,000; W.B.C. 8,700, with 68 per cent polys, 29 per cent lymphs, and 3 per cent monos. Wassermann and Kahn negative. B.U.N. 10, fasting venous sugar 75. Uranalysis revealed a faint trace of albumin, 2–4 W.B.C. and an occasional R.B.C. per high power field.

Hospital Course: On November 16, 1944, a therapeutic abortion was performed. She recovered uneventfully from this, and on November 21, 1944, was transferred from the Gynecologic Service to the Surgical Service. On November 22, 1944, the junior author began the arduous task of fulgurating the polypi in the rectum and sigmoid. The polypi were countless in number. They were both sessile and pedunculated in nature, and varied from a few millimeters to 0.5 cm. in diameter. Fulguration was performed three times a week, and after ten fulgurations the lower bowel from the anal orifice to rectosigmoid was free of polypi. She was discharged on December 12, 1944, for one month to allow the inflammation incidental to the fulgurations to subside. On January 22, 1945, she was readmitted to the hospital. Sigmoidoscopy was performed and the mucosa of the rectum and sigmoid was normal, except for several small polypi which were fulgurated. She was discharged the following day.

On January 30, 1945, she was readmitted to the hospital, and on February 5, 1945, under continuous spinal anesthesia, an end-to-side ileorectosigmoidostomy was performed. A segment of the terminal ileum was removed also at operation. On February 24, 1945, she was discharged after a satisfactory convalescence. Excellent control of bowel habit was obtained, the stools numbering two to three per day, being well formed. On April 2, 1945, patient was readmitted, and two days later a colectomy was performed down to the anastomotic site. The operation was facilitated by the fact that the small intestine was decompressed by a Miller-Abbott tube that had been inserted the day before operation. The patient developed an intestinal obstruction ten weeks after operation. An exploratory celiotomy was performed. The ileum was found obstructed, due to adhesions. The adhesions were liberated. A satisfactory convalescence occurred.

COMMENT.—This case followed our desired method of treatment from beginning to end. Patient appeared in good physical condition after the final operation. Sigmoidoscopy is to be performed at regular intervals, however.

Case 4.—E. F., a 42-year-old white male, was admitted to the Abington Memorial Hospital, March 10, 1943, with a chief complaint of constipation. For three days the patient had been having crampy, abdominal pains, vomiting, tenesmus, and inability to have a bowel movement. There was no history of weight loss, food intolerance, melena or mucus in stools or change in the bowel habit prior to the present constipation.

Past medical history was negative except for bilateral herniorrhaphy five years previously. There had been no serious illnesses in his family. No one had suffered from intestinal disorders to his knowledge.

Physical Examination: Patient was quite apprehensive. His skin was dry and he appeared somewhat dehydrated. Otherwise the examination was negative, except for slight tenderness on the left side of the abdomen. In addition, many small polypi were palpated on rectal examination.

Laboratory Data: Hb. 75 per cent, W.B.C. 8,400, with 74 per cent polys. B.U.N. was 16, chlorides 550, CO_2 62, venous sugar 86. Wassermann and Kahn negative. Uranalysis was normal. Sigmoidoscopic examination revealed many small pedunculated and sessile polypi in rectum and rectosigmoid. A barium enema revealed a filling defect involving a segment of the sigmoid at least 9 cm. in length.

Hospital Course: Five days after admission, an exploratory celiotomy was performed. The lesion seen roentgenographically was malignant, but there was no evidence of metastasis. Small polypi were present throughout the colon. The lesion was resected, a descending single-barrel colostomy performed, and the distal segment of bowel closed. After recovery from this operation, barium was injected through the colostomy, and multiple polypi were found scattered throughout the large intestine. The patient was then subjected to frequent fulgurations and the lower loop, which included the rectum and rectosigmoid was, thus, freed of polypi. On June 7, 1943, three months after

the first operation, an ileorectosigmoidostomy was performed. He was discharged three weeks following this operation. Twice he returned with signs of obstruction, each time a left pelvic abscess being present. Decompression of the intestine with a Miller-Abbott tube, and an incision and drainage of the abscess gave relief on each occasion. On March 29, 1944, approximately one year after the first operation, another exploratory celiotomy was performed, and the distal end of the ileum and the remainder of the colon was removed. Since then he has been followed in the Surgical Out-patient Department. Rectal examination now reveals evidence of recurrence.

COMMENT.—This patient had no symptoms referable to his colonic polypi and did not seek medical advice until a malignancy had occurred, with partial obstruction.

Case 5.—A white male, age 56, was admitted to the Lankenau Hospital, Philadelphia, Pa., on November 29, 1944, with a chief complaint of indigestion. Two years ago vague indigestion developed. He had experienced burning in his stomach for several years, relieved by food. Loss of appetite and easy fatigue had been present for six months. He had lost 15 pounds in six months. Three weeks ago, epigastric pain and vomiting occurred. No history of diarrhea, constipation, mucus or blood in stools.

His mother died at the age of 83, with "stomach trouble." One son was discharged from the Army because of "stomach trouble." Roentgenologic examination of his daughter revealed multiple polyposis of the colon.

Physical Examination: A middle-aged white male, extremely pallid and quite emaciated in appearance. Physical examination was negative, except for slight upper abdominal distention and several polypi palpable on rectal examination.

Laboratory Data: Hb. 31 per cent, R.B.C. 1,860,000, W.B.C., 18,500, with 75 per cent polys. Wassermann and Kahn were negative. Blood chemistry studies revealed B.U.N. 12, blood chlorides 494, serum protein 5.24, blood sugar 74, prothrombin 80 per cent. Stool examination was positive for occult blood. Uranalysis was normal.

Hospital Course: On sigmoidoscopic examination, scattered polypi were observed in the rectum. Roentgenologic studies revealed a filling defect involving the gastric antrum, and polypi in the rectum, sigmoid, transverse colon and cecum.

After frequent transfusions of blood and serum, an abdominal exploration was done. A carcinoma, involving the distal third of the stomach, was found. Likewise, polyposis of the colon existed. Subtotal gastrectomy was performed. The postoperative course was uneventful.

COMMENT.—It is extraordinary that this patient had no symptoms referable to his polypi. There seems to be an hereditary tendency to polyposis in this family, since his daughter was shown to have this disease on roentgenologic examination. There is a possibility that both his mother and son had the same condition, but we have no definite proof of this as yet.

SUMMARY

1. Five cases of multiple polyposis of the colon are reported, four of which have a definite hereditary aspect.

2. The classification, etiology, pathology, symptoms, diagnosis and treatment of congenital multiple polyposis are discussed, and special emphasis is placed on the tendency of the polypi to undergo malignant changes.

CONCLUSIONS

1. In every individual who is found to have rectal polypi, complete study of the intestinal tract is necessary. This includes proctoscopic and sigmoidoscopic examinations, barium enema studies, which include postevacuation films and double contrast studies, and, finally, roentgenograms of the stomach and small intestine.

2. The family history of every individual with multiple polyposis of the colon should be thoroughly investigated, since this condition is often on an hereditary basis.

3. Early diagnosis and treatment is very important, for intestinal polypi tend to become malignant.

4. A satisfactory plan of treatment includes fulguration of the polypi in the anus, rectum and sigmoid, followed later by ileorectosigmoidostomy and finally by colectomy.

5. The outlook for these patients is good if treatment is begun early.

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THE LOCAL USE OF SULFANILAMIDE IN THE TREATMENT OF ACUTE APPENDICITIS*

A REVIEW OF 1481 CASES

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THE PROGRESSIVE IMPROVEMENT in mortality rates in many surgical conditions has been most evident during the past five years. The reduction in mortality figures is especially marked in acute appendicitis and its complications, namely, peritoneal abscess and peritonitis.

Improvements in operative technic play only a small part since appendicectomy is a fairly standardized procedure except for the more frequent use of the McBurney incision. Some credit can be given to the advances made

CHART 1.—Made from statistics from Bureau of Vital Records and Statistics, Department of Health, City of New York.

in the use of anesthesia, and the better understanding of the problems of the surgical patient, such as fluid and chemical equilibrium and intestinal drainage. Naturally, these factors are responsible, to some degree, in lowering the mortality and morbidity figures, but, undoubtedly, the greatest factor has been the use of the sulfonamide drugs whether used locally, parenterally, or orally. It is becoming rare, indeed, for a case of peritonitis to die, unless the patient has been brought to a hospital late in an advanced stage of peritonitis. Penicillin is also proving of some value, but, since the predominating organism is *Bacillus coli,* the main reliance is placed on the sulfonamide drugs. However, all severe forms of acute appendicitis with abscess or peritonitis do best when penicillin is given in addition to the sulfonamides for its effect on the other organisms frequently present with *Bacillus coli.*

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In the majority of hospitals, the more severe cases of appendicitis and its complications are treated with sulfanilamide placed locally in the peritoneal cavity at the time of operation. In other institutions the surgeons feel this is unnecessary and give it postoperatively, by oral or parenteral routes. We feel that fewer toxic effects of the drug are seen when it is used locally. A great concentration, many times that of the blood, is advantageous in the peritoneal fluid as soon as a few hours, postoperatively. Such concentration can only be obtained in this manner. We believe that this method enables wounds to heal faster and results in fewer complications.

Sulfanilamide was first used intraperitoneally in acute appendicitis at Roosevelt Hospital January 10, 1940.[1] The results after five years are now

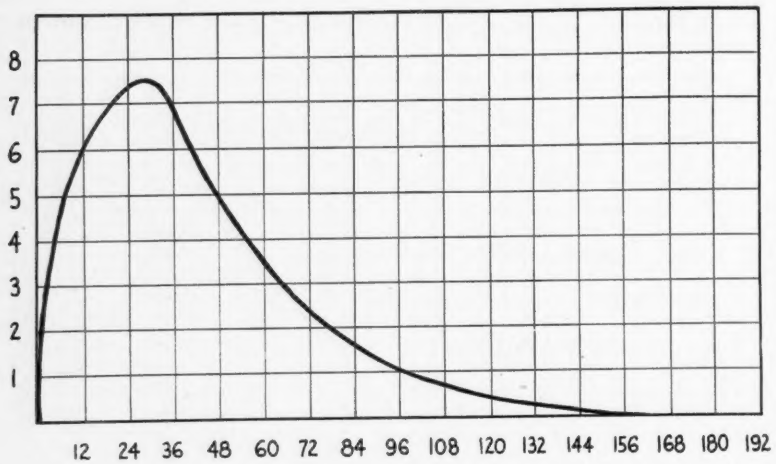

CHART 2.—Average blood level in 25 cases of peritonitis and peritoneal abscess. 6-12 grams sulfanilamide used intraperitoneally.

presented and compared with the results of the five-year period before January, 1940. In comparing the two five-year periods a simple classification of appendicitis and its complications is used. By this means we avoid the many confusing terms used in describing the types of lesions seen in appendicitis, which, ordinarily makes it difficult to accurately compare any group of appendicitis cases. The cases are classified in the following manner:

Group 1. Acute appendicitis. Here there is no gross perforation, and peritoneal fluid, if present, shows no growth. If the appendix is gangrenous, it is still placed in this group.

Group 2. Acute appendicitis with localized abscess formation.

Group 3. Acute appendicitis with peritonitis. This group includes not only

Lt. Col. James E. Thompson, M.C., A.U.S., collaborated with the author in a preliminary report on this subject J. A. M. A., **118**, 189–193, January 17, 1942. Lt. Col. Thompson has been over-seas with the Ninth Evacuation Hospital (The Roosevelt Hospital Unit) for the past three years.

the cases of spreading, diffuse, and general peritonitis, but also those of the localized variety. It is believed impossible to obtain a true knowledge of the extent of peritonitis through a McBurney incision, which was used in 96 per cent of our cases in both series. The impression would vary with the individual surgeon and could be accurately determined only after an exploratory incision. Cases classed as peritonitis must show a positive culture.

TABLE I

ACUTE APPENDICITIS SERIES: 1935-1939

Type	Cases	Deaths	Mortality Per Cent
Acute	566	3	0.53
Abscess	59	4	6.78
Peritonitis	117	14	11.96
Total	742	21	2.83

TABLE II

ACUTE APPENDICITIS SERIES: 1940-1944

Type	Cases	Deaths	Mortality Per Cent
Acute	573	0	0
Abscess	56	2	3.57
Peritonitis	110	1	0.91
Total	739	3	0.40

In both of the above series only the cases of acute appendicitis are included that were so diagnosed by the pathology laboratory. All cases of the subacute and questionable acute variety were discarded. Many cases diagnosed as acute at operation failed to be so diagnosed after section in the Pathology Department. Bacteriologic findings in the above groups of cases showed *Bacillus coli* as the predominating organism in 83 per cent of the positive cultures. Other organisms encountered as etiologic factors in order of frequency, were *gamma Streptococcus, beta Streptococcus, Staphylococcus aureus* and *albus.* Sometimes associated with *Bacillus coli,* and the other organisms, were *Bacillus lactis aerogenes* and *Bacillus mucosus capsulatus.*

TABLE III

ANALYSIS OF DEATHS: 1935-1939

	Deaths
Peritonitis	13
Pneumonia	3
Pylephlebitis and septicemia	2
Pulmonary embolism	1
Cardiac decompensation	1
Acute purulent cystitis	1
Total	21

TABLE IV

ANALYSIS OF DEATHS: 1940-1944

	Deaths
Peritonitis, shock	1
Thrombosis iliac artery, shock	1
Postoperative pneumonia, aspiration of vomitus	1
Total	3

A glance at Table III shows the cause of death in the 21 cases in the first five-year group, but, needless to say, the majority was directly due to peritonitis. In the second series, from January, 1940, through December, 1944, representing a similar five-year period, there were only three deaths in 739 cases of acute appendicitis and its complications, a mortality of 0.4 per cent. Sulfanilamide was used locally in 320 of the 739 cases, or 43.3 per cent of the total. These represented the more severe varieties of the disease. The average amount of the drug used locally in the adult cases was nine grams, two-thirds of the total amount of the drug being sprinkled in the peritoneal cavity and the remainder distributed in the wound layers. In the more severe cases of acute appendicitis without abscess or peritoneal involvement, four grams only were used, but, in the severe cases of abscess or peritonitis, we did not hesitate to use 12 grams. In those patients in whom sulfanilamide was used, the ages ranged from one year to 80 years. No definite toxic effects were seen except some cyanosis. There were no cases of leukopenia or agranulocytosis, but there were two cases of mild jaundice. In one of these cases the jaundice was felt to be definitely due to infection, as it disappeared under continued administration of sulfonamide by mouth. In 48 of the cases that received sulfanilamide locally, the administration of sulfonamide was continued orally or intravenously, usually in the form of sulfadiazine. Persistent elevation of temperature did occur in a few cases, and in some of these we felt that the sulfanilamide was directly responsible.

In this series there has been an increasing tendency toward simple drainage, without removal of the appendix, in the desperately sick peritonitis cases, unless the appendix could be removed quickly and without any search. In 20 of the above cases the appendix was not removed at the original operation. This also gave us an opportunity to observe adhesion formation at the second operation when the appendix was removed. We have been consistently impressed by the relative absence of adhesions.

Postmortem examinations were done in two of the three cases listed in Table IV. The first case was a mistake in diagnosis, and was operated upon for intestinal obstruction on the second day after admission. The patient was only five feet tall and weighed 250 pounds. At operation, through a right rectus incision, a diffuse peritonitis was found. There was an opening at the base of the appendix through which gross fecal contamination had occurred. The patient died shortly after the operation. The second case was not autopsied, but this case died two hours after an unsuccessful attempt was made to do an embolectomy. The third case died on the fifth postoperative day, at which time pneumonia and ileus were complicating the picture. A Miller-Abbott tube was being substituted for a Levine tube, which caused the patient to vomit. Aspiration occurred and the patient died in a few minutes.

The complications in the 1940–1944 series were, in general, less than half those of the earlier series. We were fortunate in not having any cardiac deaths. There was a drop of over 50 per cent in secondary peritoneal abscesses,

but we feel some of the ten secondary abscesses could have been avoided, if the cases had been drained. We deplore the tendency of not draining the peritoneal cavity merely because sulfonamide therapy is used. The old rule "when in doubt, drain" should be adhered to as it was before the use of this drug. We firmly believe that the McBurney incision should be used in all cases where a diagnosis of acute appendicitis is made. Only in very rare instances is it necessary to make a second incision for more adequate exposure.

TABLE V

COMPARISON OF IMPORTANT COMPLICATIONS

	1935-1939 (741 Cases)		1940-1944 (739 Cases)	
Type of Complication	Number	Percentage of Total Cases	Number	Percentage of Total Cases
Wound complication	85	11.5	33	4.46
Secondary peritoneal abscess	21	2.8	10	1.3
Atelectasis	16	2.1	14	1.9
Pneumonitis	12	1.6	6	0.81
Phlebitis	5	0.7	2	0.27
Pylephlebitis	2	0.27	0	0
Jaundice	1	0.13	2	0.27

In three per cent of our cases it was necessary to make a Weir extension. The McBurney incision makes for shorter operative time, quicker convalescence, fewer days in the hospital, and, in general, fewer complications. This incision was used in 96 per cent of the cases of both five-year series. It is the ideal incision for the severe peritonitis case where it is wise to leave the wound unsutured, except for the peritoneum. This cannot be done in a right rectus incision. Disruptions and postoperative herniae are not seen with a properly made McBurney incision. Drainage from a McBurney incision is better from a physiologic and anatomic standpoint, since the incision, as a rule, lies directly over, or near the pathology. Further, it is better to have the drain run from the pelvis or cecal area alongside the pelvic or lateral wall of the abdomen, rather than across loops of small intestine to emerge from a rectus incision.

A great deal of experimental work has been done in regard to the effects of sulfonamides used locally. When sulfanilamide is used properly in the correct dosage, and when it is distributed evenly throughout the wound, no interference with wound healing occurs, and the tensile strength of the wound is not diminished. We do not advocate the use of sulfathiazole and sulfadiazine locally, as we feel it is too slowly absorbed. We have used equal parts of sulfanilamide and sulfathiazole, but only in our drained peritoneal cases.

In severe peritonitis cases local anesthesia is often desirable and its use may avoid a fatal outcome. Preoperative medication with demerol and scopolamine undoubtedly aids the patient undergoing local anesthesia; frequently the patient does not remember being in the operating room. In a small number of cases it has been necessary to use small amounts of pentobarbital sodium intravenously toward the end of the operation. Use of

the oxygen positive pressure mask for 12 to 24 hours postoperatively will diminish the occurrence of atalectasis and pneumonitis and help the patient from a circulatory standpoint. The positive pressure mask, with its high concentration of oxygen, is an aid in the treatment of postoperative distention, in addition to the usual intestinal suction. Some of the more recent positive pressure masks have an opening in the apparatus which provides for the insertion of a Levine or Miller-Abbott tube.

Formerly too many of the acute appendicitis cases were operated upon too quickly after admission. There was no regard to dehydration, depleted protein, chlorides, and glucose. We do not hesitate to delay operation for three or four hours where it is necessary to supply fluid, plasma, or blood, and we believe that the better postoperative results justify the delay.

SUMMARY AND CONCLUSIONS

Sulfanilamide has been used locally in 320 out of a total of 739 cases of acute appendicitis and its complications during the past five years. The mortality in this group of cases was 0.4 per cent. The postoperative complications were relatively few.

In a similar group of cases in the five-year period before the use of local sulfanilamide, 742 cases were operated upon, with a mortality of 2.83 per cent and there were considerably more complications.

No serious toxic effects followed the use of sulfanilamide locally in the peritoneal cavity.

Local use of sulfanilamide is preferred to the use of sulfonamides orally or intravenously, because of the high local concentrations of the drug in the peritoneal cavity.

Cases complicated by abscess or peritonitis are given penicillin postoperatively in addition to the local use of sulfanilamide.

More frequent use of the McBurney incision is advised. All doubtful cases should be drained as they were before the use of the sulfonamide drugs.

The necessity of delay in operation in those cases having diminished body fluids and disturbed chemical equilibrium, until some correction of these conditions can be made. Delay should not exceed three or four hours.

More frequent use of local anesthesia is recommended for the critical peritonitis case, especially when simple drainage is the operation of choice.

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SECONDARY HEMORRHAGE ARISING FROM GUNSHOT WOUNDS OF THE PERIPHERAL BLOOD VESSELS*

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SECONDARY HEMORRHAGE following gunshot wounds was formerly a common complication. In a series of 10,000 patients with wounds involving the long bones during the last war, Waugh[1] reported an incidence of 14 per cent secondary hemorrhage during the first year and 9 per cent during the second. Although present-day methods of débridement, immobilization and chemotherapy have resulted in a striking reduction in the frequency of this complication, the occasional occurrence of severe bleeding, often with disastrous consequences, still makes this problem an important one.

The following case is illustrative of what may result from an unrecognized injury of a major blood vessel:

Case 1.—The patient, age 27, a Chinese soldier, was struck just below the left shoulder by a .25-caliber bullet on March 1, 1944. The wound of entrance was over the lateral surface of the left arm below the acromium. He suffered a compound fracture of the humerus. The bullet lodged in the soft tissues of the axilla. Débridement of the wound had been performed in the forward area, and a plaster encasement had been applied to the left shoulder with the arm in abduction. At the time of admission he was moderately anemic, Hb. 8.8 Gm. It was noted that the radial pulse was normal. There was no swelling of the hand. Sensation and motion of the fingers were present. During the first week after admission he ran no fever. On March 14, two weeks after injury, the original encasement was changed. The wound appeared clean. A hanging plaster encasement was applied to the forearm and arm for traction and, in addition, a separate spica to cover the wound. On March 17, at 0700 hours, he was seen by the Officer of the Day who noted on the patient's record: "While brushing teeth a few minutes ago, patient must have opened wound. Began to bleed under encasement. It is now stopped and condition is good. Will probably need a new encasement."

At 1000 hours, the Ward Officer noted: "No further bleeding. General condition satisfactory. The wound can be seen underneath the encasement and is dry now." That afternoon, at 1330 hours, he again bled, vomited and fainted, even though the loss of blood was small. The Ward Officer removed the encasement, but the wound was then dry. On making inquiries the corpsman stated that he had seen the patient "picking at his wound with chop sticks," and it was thought that this action had caused some bleeding from the granulations.

For the next ten days there was no further bleeding and, although he now was running a little fever, the patient's course was, in the main, uneventful. The alignment of the fracture was satisfactory.

On March 27, almost four weeks after his original injury, at 0240 hours, the patient suddenly screamed. The corpsman found him sitting up in bed in a pool of blood. While his encasement was being cut off and efforts were being made to start a transfusion the patient died.

Incidence.—During the past year secondary hemorrhage from peripheral blood vessels has taken place in only 23 cases out of a total of 2,168 patients with gunshot or shell wounds of the extremities and neck cared for in this

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Army General Hospital.* In eight of these cases the bleeding was from small vessels and was readily controlled by packing or by simple ligation of the bleeding vessel in the wound. In the remaining 15 patients, the hemorrhage resulted from the injury of a major blood vessel. It has been my privilege to examine 14 of these patients either before or after the bleeding and to operate upon 12 of them. It is on the basis of the findings in these cases that this study is made. It is hoped that by such an analysis wounds of major blood vessels may be recognized sufficiently early so that secondary hemorrhage may be avoided.

Diagnosis.—The location of the wound in every case of secondary hemorrhage was in the immediate proximity of the vessel involved. The position of the wound and the probable course of the missile should, therefore, suggest the possibility of vascular injury. In a series of 1,162 cases of secondary hemorrhage, Waugh[1] found that 68 per cent were associated with compound fractures. A similar frequency was found in our patients. Yudin,[2] in a recent series of 500 cases of compound fractures of the femur, reported that delayed hemorrhage was responsible for death in 1.6 per cent. In Table I is shown the location of the vessel responsible for the hemorrhage in this series.

The second finding of importance was the history of bleeding after injury. In two-thirds of the cases there was either a clear story of massive hemorrhage, or recurrent bleeding while in the forward area, or of severe anemia at the time of admission. Such loss of blood should make one suspect the injury of a large vessel.

Physical examination at the time of admission was not helpful. In spite of proven large lacerations of major arteries, the peripheral pulses were described as normal in six cases, diminished in two, and absent in only two patients. Of more significance was the presence of peripheral edema and a sense of numbness of the distal parts of the extremity. One or the other of these signs was present in the majority of patients. Tissue damage from severe ischemia was present in the hands of two patients, even though at the time of admission the fingers were warm and the radial pulses were palpable. Such edema of the intrinsic muscles of the hand could only have been caused by severe ischemia of long duration. Since the circulation was adequate at the time of admission, this finding pointed to a severe arterial injury for which compensation had been provided either by resumption of blood flow through the injured vessel or by the development of collateral circulation.

An hematoma about the injured vessel was present in four cases, although a systolic bruit was audible in but two of these. On the contrary, no bruit was audible in four other patients, even on careful examination with the possibility of an arterial lesion in mind. Gage[3] has stated that "sometimes the murmur does not manifest itself for hours or even days following vascular injury."

The interval of time between wounding and secondary hemorrhage was

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quite variable. During World War I,[1] bleeding most frequently occurred between the 10th and 16th day. Our earliest case bled on the second day while the longest interval was three months. Two-thirds of the patients bled within the first two weeks.

In going over our cases, we were chagrined to find that in seven of the cases, as in the patient whose clinical history is given above, one or more episodes of bleeding occurred before the hemorrhage which prompted operation. Failure to heed this "red signal" was responsible for death in only this instance, but in several other cases it was only the prompt intervention of skilled ward personnel which prevented a fatal outcome. As emphasized by Waugh,[1] "a small initial hemorrhage occurs in more than half the total cases and constitutes an inexorable indication for exploration of the wound. The nursing staff must be instructed to report even a slight hemorrhage or the discharge of small clots at once. Often, should this warning be disregarded, within a few hours there is a greater, maybe a life-endangering, loss." He goes on to comment on the constitutional reaction to the premonitory leak, which is sometimes out of all proportion to the amount of blood lost. This observation was dramatically confirmed in the first case here reported. In the majority of the patients the hemorrhage occurred spontaneously but in several there was a reasonable explanation which lulled us into a false sense of security. Mention has been made of "brushing the teeth" and "picking at the wound." Sudden movements—getting off the bed pan, falling to the floor and even refracture of the femur, could not serve as a reasonable excuse for the bleeding since in each case exploration, after a second hemorrhage, disclosed the laceration in the artery.

Since almost all compound fractures are now evacuated to the rear in plaster encasements, and since many are subsequently treated by plaster fixation, the question arose as to whether encasement of the wound in plaster contributed to the difficulty in treatment of the secondary hemorrhage. In three cases the patients bled into their encasements, and there is no doubt but that the presence of the encasement hindered early diagnosis and treatment. The incidence of bleeding is so small and the advantages of plaster fixation so well established that the added risk seems to be of little significance. But all the more emphasis should be placed on a search for signs of involvement of the major blood vessels before the encasement is applied.

Management.—Once the hemorrhage has occurred, immediate arrest of the bleeding is imperative. Packing of the wound with dry gauze and the application of pressure has generally been sufficient. Digital compression of the artery above the bleeding point may be necessary. A tourniquet about the limb should be used only as a last resort because of the damage to the peripheral tissues. Finally, especially in small wounds of the neck, digital compression of the bleeding point may be the only method which will suffice.

As soon as the bleeding has been arrested the blood lost should be replaced by transfusion, and arrangements should be made for immediate exploration of the wound. A long incision for adequate exposure is essential since the

circulation through the afferent artery must be controlled before the bleeding point can be found. We have not hesitated to open and extend the original wound. Proximal ligation through a separate wound has been performed only once, and this was for recurrent bleeding after a previous ligation of the femoral artery. After adequate exposure, rubber tubes are then placed about the artery both proximally and distally to control the bleeding. After this procedure, the bleeding point can be sought in a dry field without the danger of injury to accompanying nerves or other important structures.

U. S. Army Medical Museum A44719

Fig. 1.—Ruptured false aneurysm of popliteal artery. Femoral artery controlled with rubber tubing.

Figure 1 illustrates the control of the femoral artery in the adductor canal, with exposure of a ruptured false aneurysm.

It has long been recognized that laceration of a large vessel is more likely to lead immediately to severe bleeding than is complete severance, since in the latter case the hemorrhage is in part controlled by retraction of the two ends of the vessel. It was, therefore, not surprising to note that in every case but one in our series, a lacerated vessel was responsible for the bleeding; and even in that instance, recurrent hemorrhage which required a second operation took place from a partially divided vessel. Sir George Makins, according to Waugh,[1] believed that "an incomplete lesion of a blood vessel preëxists in every case of secondary hemorrhage."

At the time of operation, considerable difficulty may be encountered in

finding the laceration in the artery. The bleeding has presumably already been arrested on the ward by the preliminary packing and pressure. The patient's circulation is probably depressed from the loss of blood. The laceration in the vessel has again been sealed by a clot. In five of our cases, even after wide exposure, the point of bleeding was not at first apparent. With gentle manipulation, however, especially when the arterial pressure was raised by transfusion of blood, the actual source of the hemorrhage was revealed by a gush of blood. A large laceration in a major vessel has been responsible for the bleeding in all our cases. In one patient, when we thought that a small branch of the main artery was responsible for the bleeding, a second hemorrhage after transfusion forced us again to explore the wound. Large laceration of the popliteal artery was then found. In no case has it been necessary to pack the wound. This procedure has been recommended,[1] with subsequent removal of the packing in the operating theater 48 hours later, in those cases in which the site of the bleeding cannot be found.

Neck Wounds.—Severe hemorrhage from a large vein, presumably the internal jugular vein, was encountered but once.

Case 2.—The patient, a Chinese soldier, age 26, was struck on June 2 by grenade fragments over the midpoint of the right sternomastoid muscle. The fragments lodged in the body of the third cervical vertebra. Débridement of the wound was performed in the forward area. Although no operative notes accompanied the patient the fact that the wound had been packed suggested that severe bleeding had occurred. There was little swelling of the neck but the patient was hoarse and had a Horner's syndrome on the right side. Five days after injury, removal of the packing on the ward was followed by a brisk gush of venous blood. The bleeding was controlled by repacking the wound. He was taken to the operating room, local anesthesia was injected into the margins of the wound and the packing again removed. The resultant hemorrhage was definitely venous in character, and was readily controlled by digital pressure in the wound. Since the induration of the tissues and the site of the wound made exposure of the internal jugular vein hazardous it was decided to control the bleeding by suture of the wound. Five silkworm-gut sutures were placed through the skin and deeper tissues. As the middle one was tied the finger was removed from the depths of the wound. No further bleeding occurred. The patient was kept in a sitting position for one week. The wound healed well. Examination of the eyegrounds showed no venous engorgement. After healing was complete there was no bruit audible over the right side of the neck.

There were two additional patients who developed secondary hemorrhage from neck wounds.

Case 3.—A Chinese soldier sustained a shell fragment wound just below the angle of the mandible on the left side on March 18, 1944. The fragment lodged in the transverse process of the first cervical vertebra. Débridement was performed in the forward area. The note which accompanied the patient stated that "much bleeding" was encountered. He was admitted to the hospital the following day. Three days later swelling was noted below the mandible on the left side. This swelling increased in size and the patient complained of severe pain. His temperature was normal. On March 30, 12 days after his injury, with the preoperative diagnosis of cervical abscess, the mass was incised. A "rush of blood" occurred, which was controlled with difficulty by suture of the skin and a pressure dressing. On April 7 a pulsating tumor, with a palpable thrill and a continuous murmur, was present below the lobe of the left ear. The wound was

healing but there was serous fluid weeping from the skin edges. The anterior jugular vein was distended when the patient lay down but was collapsed when he sat up. The pulse rate dropped from 72 to 60 on pressure over the carotid artery sufficient to abolish the murmur. A diagnosis of arteriovenous aneurysm was made. Hemorrhage from an arteriovenous aneurysm is rare because of the free communication with the venous side of the circulation and the resultant low pressure within the aneurysmal sac. In this particular case it was considered desirable, if possible, to allow complete healing to take place before excising the fistula and, accordingly, a tight bandage was applied. In retrospect, this procedure was probably a mistake since compression of the tissues would, if anything, raise the pressure in the aneurysm through pressure on the venous outlet. The following morning, at 0530 hours, the patient cried out and the nurse found him sitting up in bed with blood streaming from his neck. She controlled the bleeding by digital pressure. The patient was taken to the operating room and, under local anesthesia, the external carotid artery was exposed below the digastricus. The circulation was temporarily occluded by a rubber tube. The arteriovenous aneurysm was then opened and excised with quadruple ligation of the component arteries and veins. It had originated from the external maxillary artery and the temperomaxillary vein.

COMMENT: This case emphasizes the importance of considering the possibility of aneurysm before incision of a mass in the neck, especially following gunshot wounds in this region. Also, it would have been wiser to have excised the aneurysm as soon as the diagnosis was made in this case. Only the prompt action of the nurse in attendance prevented this patient's death.

A second fatality in this series occurred from a wound of the carotid artery.

Case 4.—A Chinese soldier, age 24, was struck by a shell fragment on June 5. The missile entered the right side of the neck through the upper third of the sternomastoid muscle and lodged against the fifth cervical vertebra. He was admitted four days after injury. His voice was hoarse and he had a Horner's syndrome on the right side. Examination of the throat revealed paralysis of the right vocal cord and an old submucosal hemorrhage on the right side of the posterior wall of the pharynx. There was induration of the neck but no pulsation, bruit or thrill. The wound of entrance was clean. Roentgenologic examination, 11 days after injury, revealed deviation of the trachea to the left and a swelling of the retropharyngeal tissues. His course in the hospital was quite uneventful. He was ambulatory and his wound was almost healed. On July 2, four weeks after injury, he was found by the corpsman, at 2330 hours, lying on the floor in a pool of blood. He died in a few minutes.

COMMENT: In spite of the absence of pulsation, thrill or bruit, this patient probably had an extensive laceration of the carotid artery. Clearly, exploration should have been performed. The associated injuries to the vagus and cervical sympathetic nerves, together with the presence of a mass in the neck and roentgenologic evidence of retropharyngeal swelling and deviation of the trachea to the opposite side, should have been sufficient indication that arterial hemorrhage had occurred.

Gas Gangrene.—Gas gangrene was present at the time of operation in two cases.

Case 5.—An American soldier was wounded through the right thigh by a .30-caliber bullet on July 11. He sustained a compound fracture of the femur at the junction of the distal and middle thirds. Débridement of the wound and plaster fixation were not possible until 22 hours after injury. He was admitted to the hospital four hours later. At that time, he was disoriented, his temperature 103° F., and pulse 130. The toes were

warm, though edematous. Sensation was normal. The following afternoon, at 1530 hours, because of the swelling of the toes, the encasement was split. An hour later, while he was receiving a transfusion, he suddenly had a large hemorrhage. The encasement was rapidly opened and the bleeding controlled by packing. He was operated upon at 1745 hours. The posteromedial wound just above the popliteal space was explored, the femoral artery isolated and controlled in the lower part of the adductor canal. A small branch of the femoral and second small laceration in the arterial wall were thought to be responsible for the bleeding. The patient's condition was poor. He was given 1000 cc. of blood during the operation. After the dressings were applied and a Kirschner wire inserted through the tibia a second severe hemorrhage occurred. Reëxploration of the posterior wound now disclosed a large laceration in the popliteal artery. The artery was ligated and divided.

After recovery from shock, at 2000 hours, ischemia of the leg was marked and crepitation was present in the muscles on the lateral side of the leg just below the knee. The leg was amputated through the fracture site, under local anesthesia. His temperature and pulse kept rising, he became stuporous, and died four hours after operation. Culture from the wound revealed both *Cl. welchii* and *Cl. sordelli.*

COMMENT: Earlier recognition of the gas gangrene with immediate amputation at a higher level might have saved this patient's life.

Case 6.—A Chinese soldier, age 24, was wounded by a shell fragment in the right arm on March 2. The missile entered the anteromesial surface of the upper arm. The wound of exit was on the anterolateral surface at the same level. At the time of admission on March 3, his hemoglobin was only 8.0 Gm. The right arm was swollen, but the radial pulse was good. A few gas bubbles were present in the wound, but there was no crepitation in the tissues. Two days later the swelling had increased. On the fifth day after injury a moderately severe hemorrhage occurred, which was thought to be venous in character. The drain was removed since it was pressing on the brachial artery. Since it was felt that the bleeding had occurred from excessive movement, an abduction encasement was applied to the right arm. The left shoulder was incorporated in the encasement, but the injured right shoulder was left covered simply by a dressing. The following night, March 8, the patient fell from the bed pan to the floor, broke the encasement, and was found by the Officer of the Day lying in a pool of blood. The corpsman stated that the patient had been slightly irrational for some days but since he had also suffered head trauma it was to this injury that his mental confusion was attributed. Hemorrhage, clearly arterial in nature, recurred the next afternoon. The pulse at the wrist which had previously been present was now gone. In the operating room the axillary artery was first controlled and the brachial then exposed. It was found to be halfway cut in two at the junction of the upper and middle thirds of the arm. It was divided and ligated. By the following morning gas gangrene was present. Amputation through the upper third of the arm was followed by recovery.

Ischemic Gangrene.—In addition to the two cases of gas gangrene in which amputation of the extremity was performed, a third patient developed gangrene requiring amputation after ligation of the femoral artery just above the popliteal space.

Case 7.—This patient had been wounded by a shell fragment on July 11. The missile traversed the left thigh, causing a supracondylar fracture of the femur. The original bleeding had not been severe. He was admitted on July 12. On examination, the left foot was warm and the dorsalis pedis pulse was palpable. His wound became infected and he ran an intermittent fever. About four weeks after injury, pitting edema of the foot was noted. On September 15, two months after injury, bleeding took place

from the posterolateral wound. It amounted only to about 100 cc. of serosanguineous material, although some small clots were present. This hemorrhage came on immediately after use of the bed pan. Distortion of the thigh was noted. Roentgenologic examination confirmed the clinical impression of displacement at the fracture site, even though his leg had been in skeletal traction at the time. When I examined him the ankle pulses were normal, motion and sensation of the toes were present, and there was no pulsation, thrill or bruit over the femoral vessels just above the knee. One week later, an accumulation of pus on the posterolateral aspect of the lower thigh was drained. He continued to show a slight amount of blood on his dressings. The medial wound by this time was almost healed. On October 8, nearly three months after his original injury, a brisk hemorrhage occurred from the posterolateral wound. The bleeding was controlled by packing. Under spinal anesthesia, the femoral artery was exposed in the adductor canal and, after controlling the circulation, the site of the bleeding was sought. It proved to be a ruptured false aneurysm (Fig. 1). Release of the distal end of the artery allowed no "back-bleeding." It was felt that the collateral circulation was insufficient and, accordingly, the laceration in the femoral artery was sutured. No pulsation of the artery below the line of suture took place after release of the upper rubber tube. Marked ischemia of the tissues below the knee was present after operation. The lower leg was refrigerated for five days, but no improvement took place. Amputation through the fracture site was performed on October 14. Dissection of the arteries of the leg revealed that thrombosis had occurred below the point of suture.

COMMENT: In retrospect, it would have been better to have ligated and divided the artery at the time of operation. By such a procedure, progressive arterial thrombosis and spasm of the artery distal to the point of injury might have been prevented.

Suture vs. Ligation.—Suture of the laceration of the arterial wall was attempted only twice. It failed in the case cited above. It was successful in the second patient, whose brachial artery was found to have been lacerated when his wound was explored for secondary hemorrhage two weeks after his original injury. After suture of the arterial wall, pulsation of the artery distal to the injury was immediately apparent. Subsequent examination confirmed the patency of the artery. In all the remaining cases of bleeding from large arteries, the vessel was divided and ligated. The use of catgut for the ligation of major arteries had been condemned by Reid,[4] especially in the presence of wound infection, because of the likelihood of rapid disintegration of the absorbable suture material. In all of our cases No. 1 chromic catgut was used, without untoward results. The fact that the sepsis in the wounds was controlled by adequate drainage and the use of sulfonamides may have prevented subsequent weakening of the catgut or sloughing of the tissues of the vessels at the site of ligation before permanent occlusion had occurred.

Paravertebral Alcohol Injection.—Interruption of vasoconstrictor impulses by chemical section of the sympathetic nerves has been suggested by Gage and Ochsner[5] to prevent ischemic gangrene after surgical operations on major peripheral blood vessels. Following ligation of the main artery of the extremity, injection of alcohol into the region of the paravertebral sympathetic ganglia was performed in four of our cases. Repeated injections of procaine were made in an additional case. In the single instance, when the blood supply to the extremity was seriously curtailed, in which this procedure was

not employed, gangrene occurred. It is regretted that this patient was not given the benefit of prolonged vasodilatation after ligation of the femoral artery. Gangrene might have been prevented.

SUMMARY AND CONCLUSIONS

The incidence of secondary hemorrhage in 2,168 cases of gunshot wounds of the neck and extremities was 1.06 per cent. Of the 23 patients, 15 bled from wounds of major blood vessels. The present study is based on an analysis of these 15 cases.

Laceration of the arterial wall, rather than complete severance, was found in the 13 cases whose wounds were explored. There were three deaths in the group. Two patients developed gas gangrene and one of these died. Ligation of the femoral artery resulted in ischemic gangrene requiring amputation in one case. Recovery took place in the remaining patients.

TABLE I

SITE OF HEMORRHAGE IN 15 CASES

	No. of Cases
Neck:	
Internal jugular	1
Carotid	1
Arteriovenous aneurysm	1
Upper extremity:	
Axillary	2
Circumflex humeral	1
Brachial	4
Lower extremity:	
Femoral	4
Popliteal	1

A history of severe or recurrent hemorrhage in the forward area, or the presence of severe anemia on admission, is indicative of injury to a large blood vessel. Secondary hemorrhage is to be anticipated in this group. In spite of a demonstrated large opening in the main artery of the extremity, the peripheral pulse was normal in six cases, reduced in two, and absent in only two patients. Periarterial hematoma was found in four individuals, but pulsation and bruit was present in only two of these. Peripheral edema or diminished sensation in the distal parts of the extremities was present in three-quarters of the cases with lacerations of the blood vessels of the extremities.

One case is reported of the rupture of an arteriovenous aneurysm of the neck. In a second case of secondary hemorrhage from the neck, the bleeding, which was definitely venous in origin, was controlled by suture of the tissues over the bleeding point. Both cases recovered.

In all patients the major artery was exposed by wide extension of the original wound. Suture of the arterial laceration was attempted on two occasions. It was apparently successful in one case. In the second case thrombosis at the suture line extended downward and contributed to the development of gangrene. In the remaining patients the artery was divided and

ligated with No. 1 chromic catgut. No untoward results were observed after the use of this absorbable suture material.

Injection of alcohol into the region of the paravertebral sympathetic ganglia was performed in four cases, with good results. In a fifth patient, procaine was repeatedly injected.

A small initial hemorrhage occurred in seven patients before the severe blood loss which prompted operation. Attention is again[1] called to this "red signal" as an "inexorable indication for exploration of the wound."

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SECONDARY INFECTION OF WOUNDS*

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Secondary infection following the original infection of an open wound is so commonplace as to pass unnoticed. The average surgeon accepts it as a matter of course and unless the patient's life is endangered or his leg about to require immediate amputation, he thinks nothing need be done to improve the situation. Actually, secondary wound infection is the principal cause of delayed healing and impaired function. Moreover, such infections are to a great extent preventable, and even established infections can frequently be eliminated.

Primary wound infections (to distinguish them from the secondary) are those caused by bacterial contamination at the time of injury. These bacteria are derived from soil, clothing, skin and foreign bodies. The organisms existing in soil usually belong to the fecal group (*Entero-bacilli* and *Clostridia*) or, as Meleney has classified them, pathogenic aerobic gram-negative bacilli and *Clostridium welchii.* Those found in the skin and clothing are *hemolytic Streptococcus* and coagulase-positive *Staphylococcus aureus.* Hematogenous osteomyelitis, which is usually produced by the *Staphylococcus aureus,* remains an uncomplicated infection until drainage has been established and other organisms have gained access to the wound. Thereafter, the problem is to obtain healing of a wound in which there is established, mixed infection.

Secondary infections are those caused by bacteria introduced into the wound at any time after the initial injury. These invaders may come from the skin of anyone touching the wound, from unsterile inscruments or dressings or from the respiratory tract of the patient or of his attendants. They may fall into the wound with dust from the floor of a ward and may be drawn by capillary attraction through a soggy dressing which is in contact with soiled linen.

The organisms, thus acquired, usually consist of *hemolytic Streptococci* and *Staphylococci* and *Bacillus pyocyaneous.* In a recent editorial in Lancet, it was reported that a survey revealed these organisms to be present in only 5 per cent of fresh wounds upon admission to the hospital, but after a week in the institution 50 per cent of the wounds contained secondary invaders and later on they were found in 70 to 80 per cent of the open wounds. Altemeier,[1] at the Cincinnati General Hospital, found that contamination of fresh accidental wounds with *hemolytic Staphylococcus aureus* had already occurred in 35 per cent of the cases by the time débridement was done, the

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organisms having been introduced probably during the administration of first-aid or during transportation, early examination and observation prior to operation. With regard to other organisms Altemeier adds: "In previous wars as well as in time of peace the spread of *P. aeruginosa* through a crowded hospital surgical ward was well known and was easily recognized by the green or greenish-blue discoloration of the dressing. This phenomenon is of great importance since it is a visible indication of flaws in surgical technic and it would be probably easier to establish growth of a greater pathogen than *P. aeruginosa* such as the *hemolytic Streptococcus* or the *hemolytic Staphylococcus*."

TABLE I

SOFT-PART WOUNDS

Incidence of Pathogenic Aerobic Gram-negative Bacilli and *Cl. welchii* in Débrided Tissue—Persistence—Later Appearance in Cases When Not Originally Found*

	Pathogenic Aerobic Gram-negative Bacilli			*Clostridium Welchii*		
	Débrided Tissue	Persisting	New	Débrided Tissue	Persisting	New
Total	147	26	33	138	12	9
Serious infections	21	11	8	13	4	3
Trivial infections	20	8	19	19	3	4

* From subcommittee on Surgical Infections and Burns, National Research Council.

TABLE II

SOFT-PART WOUNDS

Incidence of *H. Strep.* and Coag.-pos. *Staph. Aureus* in Débrided Tissue—Persistence—Later Appearance in Cases When Not Originally Found*

	Hemolytic Streptococcus			Coagulase-positive *Staphylococcus Aureus*†		
	Débrided Tissue	Persisting	New	Débrided Tissue	Persisting	New
Total	55	11	18	74	16	51
Serious infections	9	4	6	7	3	14
Trivial infections	13	5	6	14	10	27

* From Subcommittee on Surgical Infections and Burns, National Research Council.
† This does not include other coag.-pos. micrococci.

Further evidence that secondary wound infections are both frequent and serious is included in the report of the studies made by the Committee on Infected Wounds and Burns of the National Research Council.[2] The débrided tissues from contaminated soft part wounds and compound fractures were cultured and later cultures were taken from the unhealed wounds from time to time. The following tables (Tables I–IV) taken from parts of the report show the number of cases in which the various organisms identified in the débrided tissues persisted and, in addition, the number in which the various bacteria appeared as new. In both categories the resultant infections are rated as trivial or serious.

Approximately two-thirds of the soft-part wounds and one-third of the compound fractures healed without appreciable infection. Further analysis

of these figures reveals that of 414 cultures of débrided tissue from soft-part wounds some one of the four groups of pathogens persisted in 45 instances, but they appeared as "new" in 87 cases. In 362 compound fractures the same organism appeared as "persistent" in 73 cases but as "new" in 166. Dr. Meleney, who compiled the report, expressed the opinion that some of the bacteria which appeared as "new" in subsequent wound cultures represented multiplication of organisms remaining in the wound at the time of débridement but too few in number to propagate and be identified from

TABLE III

COMPOUND FRACTURES

Incidence of *H. Strep.*, and Coag.-pos. *Staph. Aureus* in Débrided Tissue—Persistence—Later Appearance in Cases When Not Originally Found*

	Hemolytic Streptococcus			Coagulase-positive *Staphylococcus Aureus*†		
	Débrided Tissue	Persisting	New	Débrided Tissue	Persisting	New
Total	50	13	32	54	19	55
Serious infections	12	8	16	8	7	31
Trivial infections	13	2	8	9	7	11

* From Subcommittee on Surgical Infections and Burns, National Research Council.
† Does not inciude other coag.-pos micrococci.

TABLE IV

COMPOUND FRACTURES

Incidence of Pathogenic Aerobic Gram-negative Bacilli and *Cl. welchii* in Débrided Tissue—Persistence—Later Appearance in Cases When Not Originally Found*

	Pathogenic Aerobic Gram-negative Bacilli			*Clostridium Welchii*		
	Débrided Tissue	Persisting	New	Débrided Tissue	Persisting	New
Total	111	20	55	147	21	14
Serious infections	28	13	17	38	12	5
Trivial infections	11	4	31	17	2	4

* From Subcommittee on Surgical Infections and Burns, National Research Council.

the débrided tissues. It seems reasonable, however, that the majority of the bacteria appearing as "new" in subsequent wound cultures were secondary invaders. It is noteworthy that these four groups of pathogens appeared as new or secondary invaders twice as often as they persisted. It is significant also that the resultant infections are rated as serious in a little less than half the soft-part wounds with "new" invaders and considerably more than half of the same group under compound fractures.

From a study of this report the following deductions may be made:

(1) Pathogenic organisms can be eliminated in most cases by the body's defense mechanisms when these are aided by adequate surgical débridement.

(2) Pathogenic organisms persist as serious or trivial infections in 10 to 25 per cent of cases.

(3) They appear as new invaders and produce serious and trivial infections in 20 to 50 per cent of cases.

(4) Of the serious persistent infections developing in wounds, probably at least half are caused by bacteria secondarily introduced.

In view of these facts, the *"laissez faire"* attitude of surgeons toward secondary wound infections is not justifiable. Surgeons are correct in placing major emphasis upon the general condition of the patient and adequate drainage but are incorrect when they ignore the changing bacterial flora in the wound under treatment.

DIAGNOSIS

From the viewpoint of treatment, any wound not making reasonable progress toward healing from day to day may be considered as secondarily infected. The indications for the procedures necessary in treatment differ in the early and late stages. Such factors as slough, deep pockets, inadequate blood supply, scar tissue, foreign bodies, cavitation and sequestra in the bone undoubtedly predispose to propagation of the organisms principally responsible for persisting infections, and these can be judged from the gross appearance of the wound from roentgenograms of the bone. Identification of the bacterial flora, especially when gram-negative organisms predominate, is of definite assistance in selecting specific agents for systemic or local treatment. The clinical picture and bacteriologic flora differ greatly, however, in the early stage of wound healing at the end of from five to ten days and in the latter stages, after two to six months.

In the early stages, fever, edema and redness about the wound, the character and quantity of exudate intermingled with blood clots, presence of sloughing tissue or ecchymotic swollen muscle ends and absence of granulation tissue suggest incomplete débridement and the presence of pabulums favorable to the growth of anaerobes and gram-negative organisms. Smears and cultures taken from pockets in the wound will usually show numerous colonies of *B. pyocyaneous, E. Coli* and *Clostridia welchii,* together with staphylococci and streptococci.

Wounds in the later stages may not be accompanied by fever, localized swelling or redness, but the margins may have formed a scar, the granulations may be pale, edematous or sloughing, and there is apt to be a sinus leading to a cavity in the soft-parts or bone. Cultured material will usually reveal a mixture of organisms with predominance of the gram-negative type.

TREATMENT

Treatment should begin at the time of injury with preventive measures. The plan of management for the early and late stages will vary somewhat with the local condition of the wound and the bacterial flora discovered in the cultures.

Preventive Treatment.—Prevention of secondary infection and consequent delayed healing must begin with proper control of the primary infection;

hence, it must start with first-aid measures. Instead of the small, thin dressing, usually applied with strips of adhesive tape and a few turns of bandage, it would certainly be more ideal if the wounds could be covered with a massive sterile dressing, applied with pressure by masked attendants familiar with sterile technic, covered with sterile, impermeable material and immobilized in a splint for transportation. Except in cases of critical hemorrhage, this dressing should not be removed until the patient is in an operating room and can be prepared for débridement. Sedation and measures to combat shock, fluid loss and blood deficiencies should be instituted promptly. Tetanus antitoxin or "booster" doses of the toxoid should be given as indicated. Roentgenograms should be made while these are under way in most cases, but extreme care should be taken to move the patient as little as possible. Hospital efficiency should make possible performance of débridement within three hours of the time of injury.

The technic of débridement will not be described in detail since it is generally well understood. Cleansing of the skin should be meticulous, the exposure wide and removal of devitalized tissue and foreign bodies thorough. In civil practice, closure of well selected cases is most desirable, but when doubt of its advisability exists, when closure can be accomplished only with tension and when tissue damage and gross contamination are coupled with an interval of more than six hours between injury and débridement, the wound should not be sutured. Local application of bacteriostatic agents after débridement probably has no real value. Systemic administration of one of the sulfa drugs or penicillin should be started immediately after operation.

The patient's general condition should receive close attention for the next week. Adequate food, blood, fluids and penicillin should be administered. Wounds that have been closed should require no further dressing until they have healed. If the wound has been left open, it should be secondarily closed from five to ten days after operation depending upon progress of the case.

Secondary closure is an operative procedure to be undertaken in the operating room. Removal of the plaster and dressing, immobilization of the extremity and preparation of the wound and surrounding skin should be carried out as was originally done for débridement. If the appearance of the wound and the discharge indicate it, a second débridement should be done and the wound again left open, dressed and immobilized. If, however, the wound is healthy, the skin edges may be undercut a little way, the muscles drawn together and the skin approximated with interrupted sutures without tension. A massive pressure dressing, an impermeable layer and a plaster encasement should then be reapplied. This dressing can usually be retained until healing occurs. If it becomes necessary to provide drainage because of fever and abscess formation, the same procedure should be carried out again in the operating room with due care to prevent secondary invasion of the wound by new bacteria. Not until the wound has been reduced to a reasonable size, with small draining sinus, should dressings be done in the ward or room through a windowed plaster.

Dressing Technic.—In view of the origin of bacteria that become secondary invaders of wounds, it is essential to revise the dressing technic usually employed. It would be ideal to have two or three rooms reserved for this purpose and to have a special team of physicians, nurses and attendants. Patients should be moved to these rooms for dressing. Special precautions against dust-borne organisms should be observed, such as ventilation, oiled floors and bactericidal lights. In one room plasters should be cut and dressings removed and immediately discarded. The patient should then be wheeled into a second room set up as an operating room where the skin should be cleansed and draped under bactericidal lamps, the wound treated and a massive dressing applied. Final bandaging and reapplication of splints should be performed in a third room. Although such a set-up would be expensive to maintain, in a private hospital it could be made self-supporting and even profitable by charges of $15 to $25 for each dressing. Work should be scheduled for the surgeons as in the operating room. Cost would be minimized by the infrequency of dressings and the shortened period of hospitalization and disability. The use of dressing trays and carts would then be reserved for removal of stitches from clean wounds and the dressing of small surface granulating areas.

Treatment of Early Stages.—When the wound is dressed from five to ten days postoperatively, if active infection is evident as indicated by the gross appearance of, or by the cultures from, the wound, or both, secondary débridement and better drainage is indicated. Bacteriostatic agents given systemically should be continued. Secondary closure cannot be considered until the wound surfaces present a healthy appearance and sloughs have disappeared. Dressings for observation should be done infrequently and with the aseptic technic previously described.

Treatment of Late Stage (Chronic Osteomyelitis).—Chronic localized osteomyelitis is the end-result of mixed infection in bone and soft tissue caused by repeated secondary infection. Treatment must be directed first toward the patient and his body defenses. Often, the first step toward this end is to provide adequate drainage of an abscess by a short operative procedure. Adequate local preparation and absolute protection against further secondary infection must be provided from the outset. Careful inventory of the patient's defense resources should then be taken and the deficiencies made up by food, vitamins, tonics, sunshine and exercise. The condition of the bone and soft parts should be thoughtfully evaluated and an operative plan formulated. If the general condition of the patient has been sufficiently restored in advance, it should be further fortified by penicillin therapy for from three to five days and repeated, careful skin cleansings and dressings. Blood should be ready for transfusion during and after operation.

The operation accomplishes removal of all scar tissue and sufficient involucrum and bone not only to flatten cavity but to permit soft-parts to fall together. The value of this step was shown by the work of Dickson, Diveley and Kiene.[3] Whenever possible the skin should be mobilized mod-

erately so that it will approximate easily at secondary closure. Regardless of the prospect of early skin coverage, the remaining bone should be covered by muscles, even when a muscle flap must be formed and slid into position. Many wounds with a single cavity or focus in portion of bone easily covered without tension can be closed primarily. The more extensive ones should be covered with vaselined gauze and left open, and, from seven to ten days

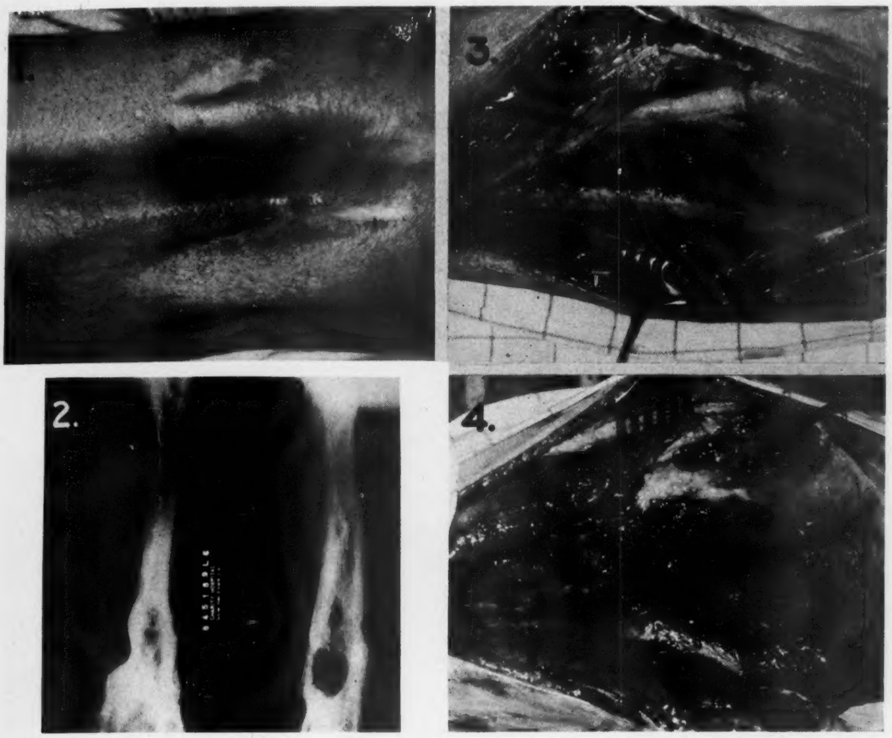

FIG. 1.—Chronic hematogenous osteomyelitis of the lower end of the femur, with established mixed infection.

(1) Lateral aspect of thigh with scar and sinus. (2) Roentgenogram showing extensive cavitation and sclerosed walls. (3) Exposure of bony cavity through lateral incision. (4) Bony cavity saucerized, biceps tendon detached and ready to be inserted into the cavity in the external condyle.

later, closed secondarily. Faultless dressing technic must be observed until the wound finally heals. Continuance of measures directed toward building body defenses is essential throughout treatment. Those with large scars and great muscular atrophy will require skin grafting as soon as the granulating bed becomes healthy.

Two case reports will suffice to illustrate the advantages of control of secondary infections by improved dressing and operative technic combined with administration of penicillin before and after operation.

ILLUSTRATIVE CASE REPORTS

Case 1.—Chronic hematogenous osteomyelitis, cavity in lower femur and condyles with ankylosis of the knee (Figs. 1 and 2).

A. A., white, male, age 35, was admitted to Charity Hospital in New Orleans on March 5, 1945.

In 1941 he had an attack of acute hematogenous osteomyelitis, with localization in the lower portion of the left femur. This was drained by a lateral incision, but the focus became chronic and has required repeated operations for drainage, sequestrectomy and saucerization. In spite of these, the process spread to involve both condyles of the femur, destroyed the knee joint and resulted in fibrous ankylosis of the knee in extension. For the past year the patient has been well enough to walk without support but with a stiff knee and a draining sinus on the outer side of his lower thigh.

On admission, physical examination revealed an active, healthy-looking man with

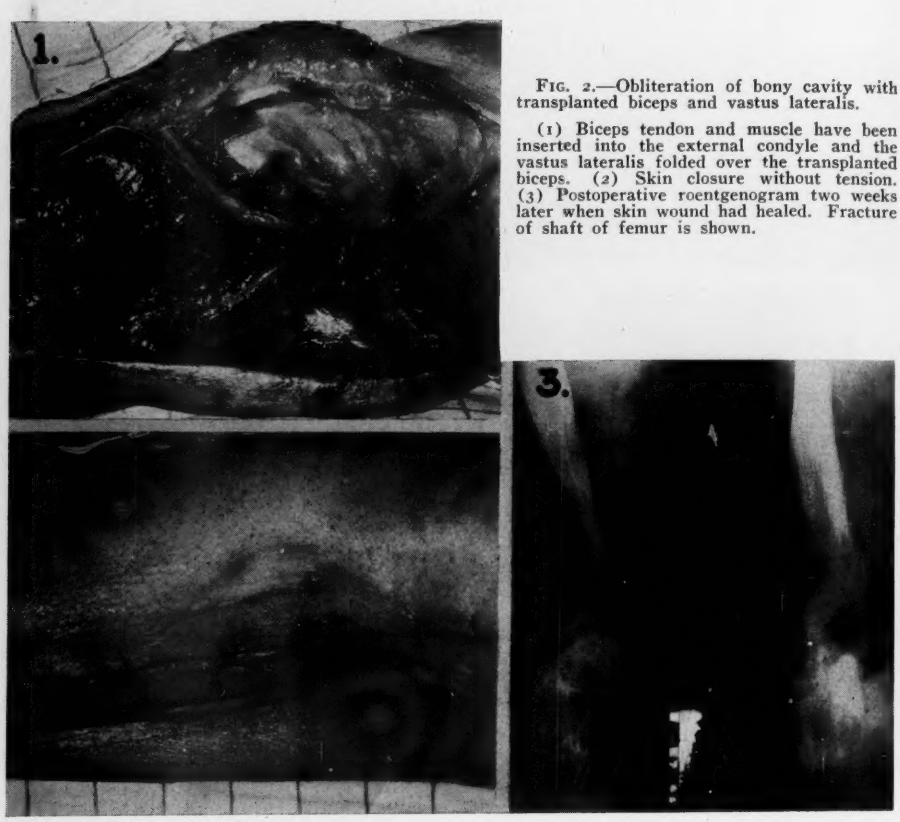

FIG. 2.—Obliteration of bony cavity with transplanted biceps and vastus lateralis.

(1) Biceps tendon and muscle have been inserted into the external condyle and the vastus lateralis folded over the transplanted biceps. (2) Skin closure without tension. (3) Postoperative roentgenogram two weeks later when skin wound had healed. Fracture of shaft of femur is shown.

no significant abnormalities other than a stiff knee and chronic osteomyelitis involving the lower half of the femur. There was a dense scar on the outer lower half of the thigh, 12 inches long, varying in width from one-half to two inches and firmly attached to the bone. Wound cultures yielded growth of *Staphylococcus aureus* and *Bacillus pyocyaneus*. A large cavity involving the lower third of the shaft and both femoral condyles could be seen in the roentgenograms. The surrounding bone was dense and sclerotic.

For seven days prior to operation, the patient received 15,000 units of penicillin every four hours and the thigh and leg were shaved and cleansed thoroughly on three successive days before operation.

Operation.—On March 13, 1945, the scar and sinus were excised down to the bone;

this left a large defect on the lateral aspect bound by the vastus externus anteriorly and the biceps posteriorly. The scar extended into the bony cavities and a central granulating cavity filled with pus was found lying in the condyles and extending into the popliteal space. All of this was removed and the bony cavity was saucerized by removal of nearly two-thirds of the circumference of the bone. Because the knee was stiff and the controlling muscles functionless, it was possible to detach the vastus lateralis from the quadriceps tendon and the biceps from the fibula and insert them into the bony cavity, thus completely filling the defect even in the condyles. The skin could then be approximated without tension and was closed without drainage. A pressure dressing and a

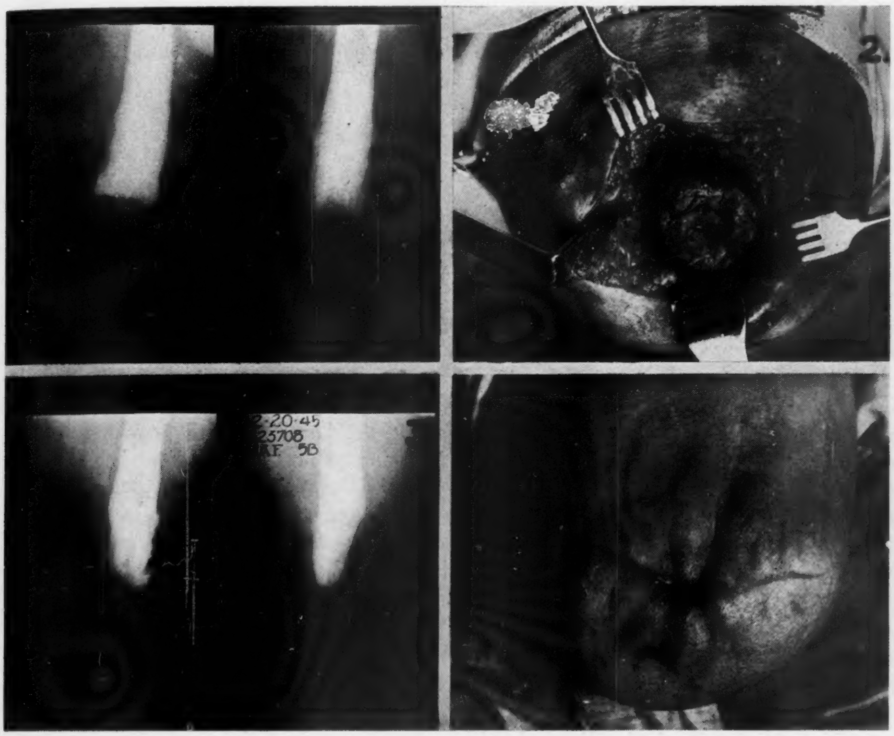

FIG. 3.—Chronic localized osteomyelitis of femoral stump.

(1) Preoperative roentgenogram showing cavitation and sequestration. (2) Scar removed exposing the end of the femoral shaft which is blocked with dense scar tissues. (3) Postoperative roentgenogram 12 weeks after operation. (4) Stump healed 12 weeks after operation.

long, leg plaster encasement were then applied. Five hundred cubic centimeters of blood were given during the operation and 15,000 units of penicillin were administered every four hours for two days. Sulfadiazine, 1 Gm. every four hours, was given for the next five days, and penicillin was again administered for the next two weeks. The systemic reaction to operation was mild, the wound healed primarily, the stitches were removed at the end of the second week, a plaster encasement was reapplied and the patient was dismissed to return in eight weeks. Postoperative roentgenograms showed a fracture, without displacement, in the upper part of the saucerized area.

COMMENT: Although the end-result in this case is problematic, the prompt healing of such a chronically infected, extensive cavity in bone can be attrib-

uted partly to the use of penicillin before and after operation, and partly to obliteration of the cavity with viable muscle.

Case 2.—Chronic localized osteomyelitis of the femoral stump (Fig. 3).

T. F., white, male, age 54, was first seen at the Ochsner Clinic, June 6, 1944, because of a chronic localized osteomyelitis in the amputated stump of his left leg. He had had an amputation through the middle third of the left femur two years before for thrombosis of the popliteal artery, which occurred as he was convalescing from pneumonia. The stump was sutured at the time of amputation, but infection developed and persisted with a draining sinus.

The patient, who appeared to be robust, was walking with the aid of crutches. Physical examination revealed generalized arteriosclerosis and heart disease. The end of the stump was edematous, the skin was glossy and mottled with purplish and brown discolorations, and there was a large granulating opening and profuse, shiny, yellowish discharge. The sinus led down to a sequestrum, seen in the roentgenograms as a large dense, cone-shaped plug at the distal end of the marrow cavity which was not entirely detached.

On July 5, 1944, the sequestrum was removed and the wound left open. At operation, it was not possible to remove all dead bone because sequestration was incomplete and the process was extending up into the marrow cavity. The wound closed gradually to a sinus, which persisted until December, 1944, when the patient was readmitted to the hospital. Cultures from the pus revealed *nonhemolytic Staphylococcus.* Penicillin was administered for one week preoperatively, and the stump was carefully cleansed and dressed.

Operation.—December 27, 1944: The scar was widely excised until healthy muscle was encountered. The fibrous plug covering the end of the bone extending up into the marrow cavity was then removed. A cavity in the bone one inch proximal to the distal end, contained several detached sequestra lying in pus. In order to avoid shortening the stump, the cavity was saucerized by bevelling the distal three inches of the shaft. The muscles were then approximated over the end of the bone and the skin was closed without tension by interrupted silkworm-gut sutures with a wick of vaselined gauze for drainage. A massive dressing and pressure bandage were applied. Penicillin therapy was resumed and continued for five days. Systemic reaction was slight. Dressings were done with aseptic technic at intervals of from five to seven days, and drainage rapidly diminished. By April 1, 1945, complete healing had occurred and the stump was shrinking satisfactorily.

Comment: Early healing of this stump is attributed to complete removal of scar tissue and sequestra, collapse of healthy muscle against the bevelled end of the shaft and penicillin therapy before and after operation. Possibly, earlier healing might have occurred if penicillin could have been given for a longer time after the last operation.

SUMMARY

Of the serious persistent infections developing in wounds, probably at least half are caused by bacteria secondarily introduced. These can frequently be prevented and even established infection can be eliminated by meticulous operative technic supplemented by the administration of sulfonamides or penicillin before and after operation. This plan of management is described and two illustrations are cited.

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TRAUMATIC SHOCK INCURABLE BY VOLUME REPLACEMENT THERAPY*

A SUMMARY OF FURTHER STUDIES INCLUDING OBSERVATIONS ON THE HEMODYNAMICS, INTERMEDIARY METABOLISM AND THERAPEUTICS OF SHOCK

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IN A PREVIOUS COMMUNICATION[1] on traumatic shock we presented evidence that the capillaries outside areas of injury do not "leak" plasma, that the essential pathology is not a disturbed capillary permeability but rather a disturbed peripheral circulation, in which the amount of blood in active circulation through capillaries is deficient. The therapeutic problem is one of restoring normal velocity and volume flow through capillaries. When the disturbance has arisen from loss of blood or plasma, the problem is solved if replacement of loss is made early enough; if replacement is made too late, the problem is not solved by blood or plasma and *a state of irreversibility exists, i.e.,* the organism progressively and rapidly deteriorates and dies even after transfusion in adequate volume is given.† It is in this phase of the process that damage which is irreparable by known modes of therapy exists. Whether this damage consists in a loss of integrity inherent in the central or peripheral circulatory apparatus *per se,* or is secondary to the failure of an extravascular controlling factor is not known.

In this communication we will present data bearing on this problem under four headings: (1) A study of the altered hemodynamics due to viscosity changes; (2) an evaluation of existing therapeutic technics applied only to the irreversible stage of shock; (3) an inquiry into certain phases of intermediary metabolism in shock; and (4) observations on the effect of viviperfusion of the liver during hemorrhagic shock.

I. THE EFFECT OF CHANGES IN BLOOD VISCOSITY UPON THE HEMODYNAMICS OF SHOCK

Although increased viscosity of the blood is properly regarded as an aggravating factor in the shock state, no precise evaluation of its importance

* The work described in this paper was done under a contract, recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and Harvard University.

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† The shock resulting from bacterial toxins, *e.g.*, of the Welch bacillus or of *B. dysenteriae* is irreversible, in the sense defined above, from the beginning. Such shock displays all the attributes of advanced traumatic shock, is not amenable to transfusion at any stage and, if specific chemotherapy is unavailing, constitutes the same therapeutic problem presented by irreversible traumatic shock.

has been made. Tourniquet shock lends itself to such a study. This condition is representative of a variety of shock states in which, because of a selective loss of plasma, hematocrit and blood viscosity increase as blood volume falls. When many investigators of this condition reported therapeutic failure following replacement of the lost plasma, an explanation for death was sought in factors not directly concerned with altered hemodynamics. Since the muscles injured by the tourniquets show marked destruction and an infectious process at autopsy, bacterial toxins or toxins derived from damaged muscle were presumed to have an etiologic rôle in the development of the fatal shock process. For reasons given elsewhere[2] we do not regard these considerations relevant and have sought an explanation for the failure of volume therapy in the hemodynamic situation. Most previous investigators had induced shock by applying tourniquets for five hours, under barbiturate anesthesia. We repeated the experiment omitting barbiturates and utilizing only morphine. In these circumstances we found the shock, if it occurred at all, to be mild and easily cured by infusion of physiologic saline solution. It was, therefore, clear that in the experiments of previous investigators the shock was precipitated and intensified by the barbiturates and that failure in therapy in large part was due to the adverse effects of barbiturates. When we applied tourniquets for 8–11 hours in unanesthetized animals, shock occurred uniformly, but it was not fatal if replacement of plasma volume loss was made in time, *i.e.*, generally before the blood pressure had dropped below 60 mm. Hg. systolic. It was evident, however, that tourniquet shock became resistant to replacement of volume loss much earlier than is the case in hemorrhagic shock. If this decreased resistance were due to absorption of toxins (the circulation through the extremities was intact throughout the experimental period so that absorption of toxins into the general circulation could occur) 5 per cent albumin solution would not have been curative at any stage, as is the case in any type of toxic shock. The effectiveness of volume replacement therapy demonstrated that no explanation for the shock state other than the hemodynamic imbalance was necessary.

Further study of the hemodynamic imbalance showed that tourniquet shock differs from hemorrhagic shock in a number of particulars as follows:

TABLE I

	Hemorrhagic Shock	Tourniquet Shock
Fluid loss into localized areas	None	Large
Blood deficiency	Whole blood	Plasma
Hematocrit	Normal or low	65-85%
Relationship of hypotensive level of blood pressure and its duration to curability by transfusion	Transfusion curative even after BP remains at 30 mm. Hg. for hours	Transfusion not curative if BP remains below 80 for hours
Capillary circulation	Slow flow and few red cells in capillaries	Slow flow, but many red cells in capillaries
Sensorium	Not dull until blood pressure well below 40	Dull at pressure below 100
Tolerance to blood sampling	Relatively good until very low pressures are reached	Poor at pressures between 80 and 100

The striking differences relate to (1) hematocrit; (2) the critical level of blood pressure; and (3) the tolerance to bleeding. It is noteworthy that successful therapy in tourniquet shock is usually accompanied by a restoration of the high hematocrit to normal or less than normal values. It, therefore, is important to see to what extent the more precarious state of tourniquet shock as compared to hemorrhagic shock can be related to the high hematocrit alone. With this purpose in mind we studied the effect of high hematocrit upon blood pressure, cardiac output and peripheral resistance, adapting Poiseuille's equation

$$\text{Flow} = \frac{\text{Pressure}}{\text{Resistance}} \times \text{Constant}$$

to a system composed of nonrigid tubes and containing a nonhomogeneous fluid, as follows:

$$\text{Cardiac output} = \frac{\text{Blood pressure}}{\text{Total peripheral resistance}} \times K$$

The total peripheral resistance is the product of the blood viscosity (n) and the resistance (R) determined by number, length and caliber of peripheral vessels. Cardiac output, blood pressure and n are determined directly. The details of the method used for measuring n and for deriving R are published elsewhere.[3] Once these values and their interdependence are known, so that a constant (K) for the normal animal can be evaluated, a standard is provided for the purpose of making comparable observations upon the dog in shock.

It was found that (1) an increase in blood viscosity has a deleterious effect upon cardiac output when the blood volume is deficient; (2) restoration of a high hematocrit to normal or below normal value improves cardiac output, but if simultaneous restoration of volume deficiency is not made, the increase in cardiac output is not enough to noticeably improve the shock state; and (3) at any level of reduced cardiac output the arterial blood pressure is higher when the blood viscosity is elevated—so that a deceptively favorable impression of the state of the circulation is given by the blood pressure reading. At blood pressure levels which are consistent with only a mild degree of hemorrhagic shock the cardiac output in tourniquet shock is already disproportionately low.

Hence, it is clear that the increased hematocrit of tourniquet shock is a liability over and above that of volume deficiency and can account for the observed differences between tourniquet shock and hemorrhagic shock. Volume deficiency is by far the greater danger and its restoration is a more urgent need than is that of normal viscosity. If an abnormally high viscosity exists in shock and is not treated effectively, the restoration and maintenance of normal blood volume becomes all the more urgent. The development of irreversibility to transfusion and the poor tolerance to bleeding in tourniquet shock at higher levels of blood pressure than in hemorrhagic shock is also explained by these findings.

II. THERAPY OF HEMORRHAGIC SHOCK IRREVERSIBLE TO TRANSFUSION

While the foregoing studies elucidate some of the hemodynamic mechanisms operating in certain circumstances, none of the observed changes in the hemodynamic equilibrium explains the failure of volume replacement to effect recovery after a period of time at pressures below 60.

Certain investigators[4, 5] insist that there is no time before death when restoration of adequate capillary flow cannot be achieved if only a sufficient amount of the right kind of fluid is given. Other agents also have been claimed to be effective even in late shock. It is necessary to be clear as to what we mean by late or so-called irreversible shock: It is that phase of shock which shows no sustained favorable response to the reinfusion of all the blood or plasma removed to induce the shock state. So long as no reliable method for confidently predicting the response to blood or blood substitute alone has been developed, claims made for the therapeutic value of any procedure or any substance other than blood or a blood substitute can be valid if effective alone and without restoring blood volume deficiency or if effective after an adequate transfusion has been shown to have failed. If the estimate of an agent's value is based upon its presumed effectiveness when given just before or along with blood or a blood substitute, unequivocal evidence must be provided to show that the latter alone would not have achieved the same result.

The following agents were studied with the foregoing considerations in mind: (1) Saline solution in large volume. (2) Whole blood. (3) Five per cent and 25 per cent albumin solution. (4) Alkali. (5) Pressor agents. (6) Succinic acid. (7) Coramine. (8) Various combinations of the foregoing agents.

Utilizing hemorrhagic shock and eliminating as far as possible operative manipulations, anesthetics and other forms of trauma, which facilitate the development of irreversibility or otherwise complicate the shock state, the agents listed were studied in every instance only after the shock state was found to be unresponsive to the replacement of all shed blood. The following results, discussed in detail elsewhere,[6] were obtained:

1. Massive infusions of physiologic saline solution may cause transitory improvement in circulation, but do not cure hemorrhagic shock irreversible to transfusion.

2. Massive infusions of isotonic (5 per cent) bovine albumin solution* greatly increase the blood volume and may sustain the circulation for a time, but only rarely result in recovery. A marked bleeding tendency is produced by this therapy. Concentrated (25 per cent) bovine albumin solution in equivalent or greater protein content is of no benefit, even if supplemented by saline solution.

* The crystallized bovine serum albumin employed in this work was prepared at the Armour Laboratories, Chicago, Illinois, by the method of Cohn and Hughes, under a contract, recommended by the Committee on Medical Research, of the Office of Scientific Research and Development.

3. Large volume intravenous infusion therapy, using either physiologic saline solution alone or albumin in physiologic saline solution, is harmful by producing marked edema of tissues, pulmonary edema, serous effusions, venous distention and widespread hemorrhage from small vessels.

4. Pitressin with or without ergotamine is of no value. A combination of pitressin with 5 per cent albumin solution is not beneficial.

5. Paredrine* (p - hydroxy - a - methylphenylethylamine hydrobromide) causes an elevation of the arterial and venous blood pressure. Venous sampling from various areas showed no evidence of preferential improvement of flow into such vital areas as the brain and liver. No improvement in cardiac output, oxygen content of venous blood or capillary flow was noted. The duration of the pressor effect is limited by the rapid development of unresponsiveness to the drug and survival time is not prolonged.

6. Hypertensin, a product of the constant interaction of renin, of renal origin, and hypertensinogen, of hepatic origin, when continuously administered, causes an elevation of blood pressure, an increase in the volume and velocity flow through capillaries, a rise in O_2 saturation of venous blood toward normal and a rise in cardiac output. Unfortunately, the dog in shock becomes increasingly tachyphylactic to this as to other drugs, so that increasing doses must be given to obtain a continuing effect. The amount required to sustain one dog for an hour or two is enormous in terms of the labor and material involved in the present methods of preparation of hypertensin. This substance is of academic rather than of practical interest at present—for even when enough material was available for a few experiments, responsiveness to it was not sustained long enough to reverse the shock process.

7. Coramine (pyridine-beta-carboxylic acid diethylamide) increases skeletal muscle tone, but does not favorably influence the course of events.

8. The correction of acidosis by the administration of sodium bicarbonate with, or subsequent to, the initial transfusion does not alter the deteriorating trend of advanced hemorrhagic shock.

9. Sodium succinate is of no benefit in the therapy of advanced hemorrhagic shock.

10. Tuamine, given when the initial transfusion is failing, causes a transitory rise in blood pressure. Survival time is not prolonged.

11. Potassium phosphate† intracisternally does not alter the deteriorating trend of hemorrhagic shock and at the same time produces undesirable cerebral excitatory phenomena.

The completely negative value of such a large variety of agents is at variance with the claims made by various investigators. Allen,[4] and Warren, *et al.*,[5] presented data to the effect that the deteriorating trend in shock in any

* Most vasopressor drugs are considered deleterious because they add to the already increased vasoconstriction with resulting further depletion of capillary flow. Since most of these drugs exert metabolic effects as well, which paredrine does not, this drug was studied extensively.[7]

† This drug, suggested by Russian investigators, was provided through the courtesy of Dr. Baird Hastings, Department of Biochemistry, Harvard Medical School, Boston, Mass.

stage can be halted or even reversed by intravenous physiologic saline solution given in sufficient volume for a sufficient length of time, with care to avoid fatal pulmonary edema. Survival was explained as due to the creation of a sufficiently high interstitial pressure so as to reverse the flow of fluids from out to into the circulatory bed. The therapeutic value of saline solution is not confirmed by our data on hemorrhagic shock, even though the volume of saline solution was adequate to achieve the tissue pressures demanded by such a postulate.

Evidence of recovery from shock includes a sustained rise in cardiac output, in acceleration of capillary flow, in oxygen content of the mixed venous blood and in blood pressure. Such changes, readily achieved during the early shock phase by transfusion, do not persist following the infusion of large volumes of fluid of any kind if given after the initial transfusion has failed. The futility of infusion of relatively huge volumes of fluids which do not escape or escape only slowly from the circulation (*e.g.*, albumin solution) demonstrates that even when there is no significant continuing loss of fluid, as in irreversible hemorrhagic shock, conditions exist that are no longer amenable to the restoration and successful maintenance of a normal or more than normal blood volume.

The infusion of large amounts of albumin or whole blood, with Ringer's solution containing alkali, is not only not effective in hemorrhagic shock, but leads to pulmonary edema and to considerable bleeding in open wounds and in the intestine. Moyer, *et al.*,[8] recommended the clinical use of physiologic saline and soda bicarbonate solution plus defibrinated blood in the treatment of burns. The recommendation may be classified with those of Fox,[9] and of Rosenthal,[10] who report a favorable response of burned patients and of rats respectively to electrolyte solutions. Since the data offered by these authors do not demonstrate conclusively that the acute hemodynamic imbalance of shock existed, any inference made, or implied, that these therapeutic measures are of value for the treatment of advanced traumatic shock from any cause is unjustified.

Wiggers and Werle[11] believe that declining myocardial efficiency is to some extent responsible for the progressive decline in the late shock phase. Recently, Page[12] identified, by cardiometric observations on the exposed heart, a measure of cardiac weakness which was not reversible by any known methods of therapy except certain aliphatic amines (one-amine, tuamine, *etc.*) with specific myocardial stimulating qualities. In six experiments with our technic of determining irreversibility, tuamine produced no rise in cardiac output or other beneficial response except a transient rise in systolic pressure. The effect of tuamine was analogous to that of paredrine. Our experiments differed from those of Page in that he gave the drug with, or immediately after, transfusion, whereas, we withheld the drug until the response to transfusion alone had been found ineffective.

Henderson[13] attributes the fault in the circulation in shock to a loss of skeletal muscle tone, with consequent failure of venous pressure and return flow. In our experiments with coramine, which increases skeletal muscle tone

to the point of extreme rigidity, blood flow was not improved. A more desirable physiologic technic to test the Henderson hypothesis would provide for alternating contraction and relaxation of muscle.

Chambers, *et al.*,[14] believe that the primary deficiency lies in the collapse of the contractile power (vasomotion) of arterioles and venules, which in late shock lose their reactivity to adrenalin. They report a favorable response[15] from the use of pitressin, with and without ergotamine, given with, and subsequent to, transfusion. In our experiments these agents were given without transfusion or after the transfusion was shown to be ineffective. No benefit was observed.

Uncompensated acidosis has long been known to persist in the untreated late shock phase and the alkali therapy introduced by Cannon[16] during World War I has recently been recommended by Levine, *et al.*,[17] who attribute to sodium bicarbonate the capacity to reverse and to cure a degree of shock not amenable to transfusion alone. They accept as a test for such a degree of shock not the fact of a futile transfusion already given, but a CO_2 combining capacity of 15 volumes per cent or less. In our experiments, when alkali was given to correct acidosis and to maintain a normal or higher than normal CO_2 combining capacity thereafter, the deteriorating trend following transfusion was not noticeably alleviated.

The tolerance of the organism to hemorrhage, when the latter is induced by the withdrawal of blood by simple needle puncture of a large artery in the intact unanesthetized and unimmobilized dog, contrasts sharply with that of the dog anesthetized, immobilized, cannulated and otherwise traumatized. This points out the need of excluding as far as possible all extraneous factors, if the shock due to volume loss alone is to be properly evaluated.* The more such factors are eliminated, the more difficult it becomes to produce irreversibility to transfusion, which then is achieved by prolonging the time and increasing the degree of hypotension. Our experiments approached the ideal of simplification only very approximately, for we used morphine and open wounds and a considerable degree of immobilization in order to obtain essential data not otherwise possible. The factor of sepsis is of dubious import, because of the absence of pathologic evidence of its presence in these experiments, and because the experimental period seems too short for the production of significant amounts of bacterial toxin. Even if bacteremia is common in these circumstances, it remains to be shown that the kind of organisms, their number and

* Experiments reported by Schachter (Am. J. Physiol., 143, 552, 1945) are a case in point. He reports that cholinesterase cures traumatic shock induced by intestinal manipulation in anesthetized hyperthyroid dogs. From his hematocrit data there is reason to believe that the plasma volume loss would not have been sufficient to induce fatal shock in unanesthetized nonhyperthyroid dogs. Presumably shock was precipitated by the superimposed aggravating factors of barbiturate anesthesia and hyperthyroidism. Plasma volume therapy failed in such dogs for the same reason that it fails in five-hour tourniquet shock induced under barbiturate anesthesia. The favorable action of cholinesterase in these experiments may have been exerted upon the accessory aggravating factors and does not justify a conclusion that cholinesterase is of value in the treatment of shock *per se*.

virulence, were of sufficient consequence during the period of experimentation to have influenced the results. Reduced to the simplest conditions it is possible to achieve experimentally, we believe that the course of events in hemorrhagic shock leading to the development of a state of irreversibility to transfusion is a function of the severity and duration of inadequate capillary flow, with a resulting cumulative adverse effect on the integrity of cellular function in general or on a basic biochemical function in a controlling vital organ such as the liver.

III. INTERMEDIARY METABOLISM IN SHOCK

Ample evidence is available of serious disturbances in kidney, liver and intestinal function[18] and of extensive biochemical abnormalities, especially in intermediary carbohydrate metabolism.[19]

Phosphorylation of enzymes essential in carbohydrate breakdown and resynthesis is seriously affected in shock.[20, 21] The lack of substrates normally available for energy release has led to efforts to replace them by others present in tissues but not necessarily serving this purpose, *e.g.*, succinic acid.*

Van Slyke[24] has succeeded in defining the limits of anoxia which the kidney can endure before irreparable damage to function results. But the effects of loss of kidney function, while capable of causing death within a few days, are not, as far as is known, operative within the shock period.† Collapse of liver function might well result in irretrievable effects and explain death within the shock period. A measure of such collapse might be reflected in aberrations from the normal sequence of reactions in intermediary metabolism.

Our primary purpose in undertaking studies of the intermediary metabolism in shock was to discover, if possible, some point in the process of biochemic disintegration, *i.e.*, failure of completion of a normal biochemic reaction, which would identify the onset of irreversibility. Thus, a tolerance test to intravenously injected carbohydrate or protein intermediates might disclose a time of crucial metabolic collapse paralleling the onset of irreversi-

* When Shorr, *et al.*,[22] showed that this substance *in vitro* increases oxygen consumption of tissue slices at low oxygen tensions, Mylon and Winternitz,[23] and Levine, *et al.*,[17] reported substantial improvement in the shock state as a result of its administration. We were unable to substantiate these claims in tourniquet shock[2] or in hemorrhagic shock.[6] Recently, Shorr,[22] and Meyer and Potter,[20] have found that the increased oxygen consumption at low oxygen tensions produced by succinic acid is at the expense of the tissue needs for normal function and to this extent succinic acid can be regarded as deleterious rather than helpful.

† Anuria resulting from organic disturbance of the kidneys is a common experience in military surgery following extensive wounds. Frequent reports from the European Theater of War refer to such patients as responding well to the initial therapy for shock, only to die later in uremia. The kidneys show casts in the tubules or cloudy swelling, or both, and no known treatment has been of any value. For such cases and for patients in shock who show deficient renal function because of prolonged ineffectively treated periods of hypotension, it is desirable, if possible, to develop a temporary substitute for kidney function, so that uremic death may not result during the time necessary for recovery of kidney function. Experiments to be reported[25] show that continuous peritoneal irrigation for days in nephrectomized dogs, before and after the development of uremia, is capable of preventing or removing the accumulation of nitrogenous substances in the blood.

bility. For this purpose we performed tolerance tests for glucose, lactic acid, pyruvic acid and a variety of amino-acids in various phases of shock. The accumulation of the latter three normal metabolites in the blood in shock is progressive.

Engel, *et al.*,[19] state that the increasing amino-acid level is an index of failure of deamination by the liver. Our tolerance curve studies showed (1) that all these metabolites, including the amino-acids, are catabolized as readily in the irreversible as in the reversible phase of shock, indeed, almost as well as in the normal animal; and (2) the increased concentration of these substances in the blood represents an adjustment of metabolic reactions at a new level. The detailed data in support of these conclusions will appear in a subsequent paper.[26]

IV. VIVIPERFUSION OF THE LIVER

Failing in our effort to correlate the development of irreversibility to a serious disruption in these processes, we proceeded to an investigation of the liver as a likely example of a vital organ whose controlling influence, if lost, may constitute the primary cause of death. There is evidence of severe liver injury in shock: (1) This organ receives much of its blood supply from the portal vein, which in shock has a much reduced volume flow of blood with a very low oxygen content. (2) Excretion of bromsulfalein is much reduced in shock. (3) The duration of survival following total hepatectomy is comparable to that following the onset of hemorrhagic shock and the blood chemistry of the two conditions is not dissimilar.[27] (4) The liver is soft, friable and discolored at death from hemorrhagic shock.

Although the rôle of the liver is still being actively studied, certain preliminary results of interest may be referred to herewith. Our approach to this problem consisted in an experimental set-up aimed at preserving the integrity of the liver,[28] while the remainder of the organism is exposed to the general effects of the shock state. This was carried out as follows: The splenic vein of a healthy dog is prepared for cannulation. Hemorrhagic shock is induced. One carotid artery of a donor dog is connected to the splenic vein of the dog in shock. Both femoral arteries of the dog in shock are connected to one femoral vein of the donor. In the delivery and return circuits calibrated manometers, constructed on the Bernouille principle, are introduced. Both dogs and the entire connecting system are heparinized. The dog in shock has been bled and when the blood pressure has been stabilized at a level of some 30 mm. Hg. all circuits are opened and the shocked dog receives through the splenic vein some 350–450 cc. of arterial blood per minute* from the donor dog. Volume flow to and return from the dog in shock is controllable and is readily equalized by adjustable clamps on the connecting tubes, so that over a period of hours no measurable difference between volume delivery and return is observed.

Experience has demonstrated that nearly all dogs in hemorrhagic shock,

* This volume flow of blood approximates the portal flow through the liver in a normal dog.[27]

maintained at a pressure of 30 mm. Hg. for from 1.5–3 hours, will be unresponsive to transfusion. Such dogs connected to an elevated bottle containing the shed blood will begin to take blood back from this reservoir as they approach the state of rapid collapse. When they have taken from one-third to one-half of the shed blood volume, well over 80 per cent will show no sustained response following the rapid reinfusion of the remainder. This "taking-up" process therefore may be used as probable evidence of the onset of irreversibility. In the viviperfusion experiments the dog in shock, as soon as its blood pressure begins to give way, "takes-up" from the donor dog, which, like the elevated bottle, acts as a supply reservoir. This is registered in terms of a decline in donor blood pressure and is measured approximately by noting the amount of blood taken in advance from a third dog which is required to return the donor blood pressure to its original level. When one-third to one-half of the shocked dog's shed blood volume has been taken up, the dog is regarded as probably irreversible. At this point the cross-circuits are closed and disconnected and the dog in shock is then given back all the shed blood. Nothing further is done. Posttransfusion observations are made and survival time observed. The same technic was used on a control group of dogs in which the jugular instead of the splenic vein was used as the route for viviperfusion.

All but one of 15 dogs in the control group died as soon as and with the same pathologic manifestations as dogs in irreversible hemorrhagic shock which are not cross-circulated. All but one of 12 dogs whose livers were perfused survived. Immediately after transfusion they were active, able to stand up and drink water. They showed no subsequent evidence of renal damage.

The uniformity of the results in each of the two groups establishes the importance of the liver in the shock process and the significance of the damage which it suffers after several hours of reduced volume and velocity of flow of anoxic blood. By preventing liver damage, irreversibility to transfusion is prevented. It is, therefore, not necessary to postulate widespread irreparable damage to cellular function in order to account for the onset of irreversibility to transfusion. Whether damage to the liver, if allowed to occur, is reversible will be determined by beginning the viviperfusion only after transfusion has been shown to have failed.

The prevention of the development of irreversibility by viviperfusion of the liver is not due to the removal of toxins or to the provision of protective substances by the donor dog during the shock phase, because the same processes operate in the dog viviperfused *via* the jugular vein.

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TRANSLOCATION OF FLUID PRODUCED BY THE INTRAVENOUS ADMINISTRATION OF ISOTONIC SALT SOLUTIONS IN MAN POSTOPERATIVELY*

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DURING THE PAST DECADE several papers from this clinic[1, 2, 3] have pointed out certain possible, undesirable complications attendant upon the unwise use of intravenous isotonic sodium chloride solution. It has been emphasized[3] that these dangers are increased many fold during the immediate postoperative period of the sick surgical patient. This concept is neither new nor original, since many authors[4] have directed attention to the potential toxicity of so-called "physiologic saline," especially when large amounts are administered.

Studies to determine the manner in which the human body handles large intravenous infusions of "salt" solutions were undertaken in patients undergoing combined abdominoperineal resections for carcinoma of the rectum. An explanation of the "salt intolerance" was sought in the excretions and retentions of sodium, chloride and water in the 30-hour period beginning with the operation. Solutions of various composition and tonicity were infused at regular intervals during this period.

The study was divided into five six-hour periods. The initial infusion was started at the beginning of the operation and the rate of administration was determined by the exigencies of the operation. The second infusion was started six hours after the first infusion. This, and subsequent infusions, were administered at approximate rates of 250 Ml. per hour for the smaller amounts and 400 Ml. per hour for the larger volumes.

Weighed dressings covered with oiled silk were applied to the anterior and posterior wounds. These dressings were changed and reweighed every 12 hours (three determinations of losses from wound drainage). In the balance studies, such losses were considered as an ultrafiltrate of plasma, since loss of blood was visibly small.

Urine was collected during the five periods and preserved with thymol and refrigeration. Two samples of blood, one with heparin and one under oil, were withdrawn before operation, at the end of operation, and at the end of the final infusion period. The urines were analyzed for specific gravity, p_H by glass electrode, potassium by the method of Fiske and Litarczek,[5] sodium by the method of Butler and Tuthill,[6] ammonia by the method of Folin and Bell,[7] chloride by the method of Logan,[8] sulfate by the method of Fiske,[10] phosphate by the method of Fiske and Subbarow,[10] total nitrogen by

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the micro-kjeldahl method of Pregl,[11] urea,[12] and carbon dioxide[13] by the methods of Van Slyke, and creatinin by the method of Popper.[14] Heparinized whole blood was analyzed for hematocrit by use of capillary tubes, for specific gravity by the method of Barbour and Hamilton,[15] and for hemoglobin by the photo-electric method of Evelyn.[16] Serum proteins were calculated from the specific gravity of the sera according to the formula of Weech.[17] Serum was analyzed for sodium, carbon dioxide content and chloride.

Blood loss during operation was estimated, and in almost all cases found to correspond to the average figures previously reported by this clinic.[18]

Procedure and Method of Calculation: The patients who served as subjects were selected only in that they were determined to be free of gross cardiovascular and kidney disease. They were prepared for operation by sulfasuccidine and Miles' regimen. If the hemoglobin was below 80 per cent, they were transfused before operation. The preanesthetic medication consisted of morphine in combination with barbiturates. Operations were performed under spinal anesthesia with either nupercaine or continuous procaine. Preoperatively, an indwelling catheter was introduced, which remained in place throughout the study.

All intake was by the intravenous route. The first five cases received 3,750 Ml. of 0.9 per cent NaCl in five equal infusions during the first 30 hours after operation. Three patients received 3,750 Ml. of 0.75 per cent NaCl plus 0.22 per cent $NaHCO_3$, an adjusted salt solution containing a physiologic amount of $NaHCO_3$ (26 mEq. per liter). One patient received 5,625 Ml. of 0.6 per cent NaCl, a solution physiologic with respect to chloride. Three patients received 7,500 Ml. of 0.45 per cent NaCl, and one patient, 7,500 Ml. of 0.375 per cent NaCl plus 0.11 per cent $NaHCO_3$.

In-put of sodium was kept constant at 578 mEq. except in the case of one patient, a small individual who received 80 per cent of the established infusion. Since the nutritional status of these patients with neoplastic disease could only be judged by body weight, and the preformed water contained in the water evaporated from lungs and skin and given off in the urine could only be guessed at, this preformed water has been ignored in the calculations of load of water. However, in Benedict's fasting subject, Levanzin, preformed water amounted to 585 Gm. during the first 24 hours of the fast.[19]

Water gained as a result of the breakdown of body protein was calculated from the urinary nitrogen by use of the factor 6.25 and the equation: water = 0.27 liters per 100 Gm. protein.[20] Insensible loss was estimated for all patients as 0.07 per cent of body weight per hour, as suggested by Adolph.[21] This value is based upon studies of normal individuals and in all probability is too conservative an estimate of losses by patients immediately postoperatively.

Water balance, thus, becomes in-put plus oxidative gain less the combined losses by way of the urine, wounds, lungs and skin. Sodium and chloride balances are calculated from in-put less urinary and wound losses.

Results: Table I summarizes the changes in the blood and serum of eight

patients receiving the five types of infusion. There is evidence that all patients handled the infusions fairly efficiently with the probable exception of Patient St. The best examples of this efficiency are the small changes produced in the concentrations of sodium and chloride in the sera of the patients receiving large infusions of hypotonic solutions. Serum proteins are lowered in every case, regardless of the volume of solution infused.

Table II presents the urinary concentrations of potassium, sodium, chloride, and the ratios of sodium to chloride of all patients during the first 24 hours

TABLE I

CHANGES IN THE BLOOD AND SERUM OF EIGHT PATIENTS RECEIVING THE VARIOUS INFUSIONS

		Blood			Serum			
Patient—Infusion		Hematocrit Vol. %	Hemoglobin Gm. %	Specific Gravity	Protein Gm. %	Carbon Dioxide* Vol. %	Chloride mEq./l.	Sodium mEq./l.
Br ♂ 0.9% NaCl	Preop.	43.0	14.9	1.0556	6.12	60.1	101.3	137.7
	Postop.	40.0	12.4	1.0476	5.27	57.4	103.2	132.5
	30 hrs.	37.3	11.6	1.0438	3.35	48.9	108.7	136.8
St ♀ 0.75% NaCl+ 0.22% $NaHCO_3$	Preop.	40.5	12.6	1.0501	6.19	58.4	104.2	136.9
	Postop.	17.5	6.0	1.0283	3.20	55.2	115.4	143.8
	33 hrs.	16.2	6.8	1.0363	5.21	58.9	105.0	136.9
	Transfus.	25.3	8.2	1.0397	5.24	63.3	106.7	136.9
Vo ♂ 0.75% NaCl+ 0.22% $NaHCO_3$	Preop.	43.9	14.2	1.0524	5.44	68.9	102.7	139.7
	Postop.	51.5	15.4	1.0551	5.62	71.8	103.0	140.6
	30 hrs.	40.0	12.4	1.0494	5.03	64.0	104.3	140.6
Mo ♂ 0.6% NaCl	Preop.	43.4	14.6	1.0546	6.50	64.2	100.8	132.3
	Postop.	44.4	13.7	1.0521	5.85	58.8	102.7	130.6
	30 hrs.	36.9	12.1	1.0473	4.96	55.1	102.5	130.4
Hu ♂ 0.38% NaCl+ 0.11% $NaHCO_3$	Preop.	50.7	15.7	1.0559	6.19	62.6	97.8	132.8
	Postop.	44.8	13.5	1.0486	4.90	58.7	99.9	132.6
	30 hrs.	42.0	12.7	1.0486	4.76	56.5	99.9	126.7
Le ♂ 0.45% NaCl	Preop.	48.9	15.4	1.0585	6.25	63.8	102.3	138.3
	Postop.	47.0	14.2	1.0571	6.12	53.2	102.0	137.2
	30 hrs.	33.3	10.5	1.0435	4.86	53.5	95.8	120.8
Sm ♀† 0.45% NaCl	Preop.	47.3	15.2	1.0582	7.01	56.5	100.7	132.6
	Postop.	42.8	13.0	1.0512	5.58	59.3	101.7	134.3
	30 hrs.	45.9	12.5	1.0501	5.11	53.8	94.8	127.1
Re ♂ 0.45% NaCl	Preop.	50.5	16.4	1.0607	6.63	57.7	103.3	135.4
	Postop.	49.2	15.2	1.0507	6.50	66.7	101.0	138.2
	30 hrs.	28.3	9.5	1.0422	4.63	53.2	99.4	131.2

* Content.
† Patient Sm received 80% of the infusion.

postoperatively. It is noteworthy that the two patients receiving the adjusted salt solution and the hypotonic, adjusted salt solution were the only subjects who excreted sodium and chloride in ratios approximating that of an ultrafiltrate of plasma.

Table III summarizes the loads of sodium, chloride and water at the end of 30 hours' infusion for eight patients receiving the various infusions.

The complete data, except for blood changes, are presented in Figures 1 through 6. Loads of water, sodium and chloride, and urinary losses of sodium, chloride, potassium, phosphate, sulfate and nitrogen are charted as cumulative balances and losses throughout the 30-hour period. The nitrogen scale has been adjusted to correspond to the nitrogen to potassium ratio existing in tissue. As a result, Figures 1 to 6 stress the presence of "excess

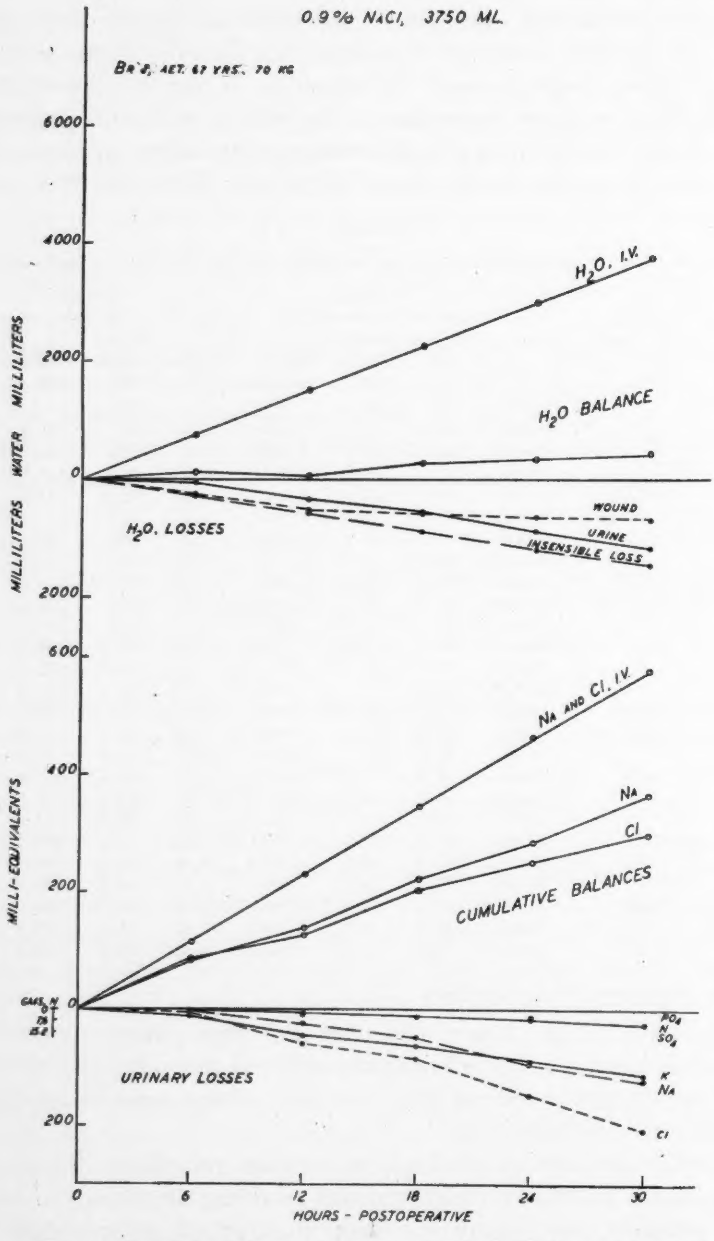

FIG. 1.—Cumulative balances of water, sodium and chloride, and losses of water and urinary constituents of patient Br., m., No. 565,142, who received 3750 Ml of 0.9% NaCl in five equal infusions during 30 hours following combined abdominoperineal resection for carcinoma of the rectum. The patient was not prepared for operation by Mile's regimen because of a cecostomy performed 19 days before. Operation was performed under continuous procaine spinal. Postoperative course was uneventful, except for a mild, transitory hypotension lasting for six hours after operation.

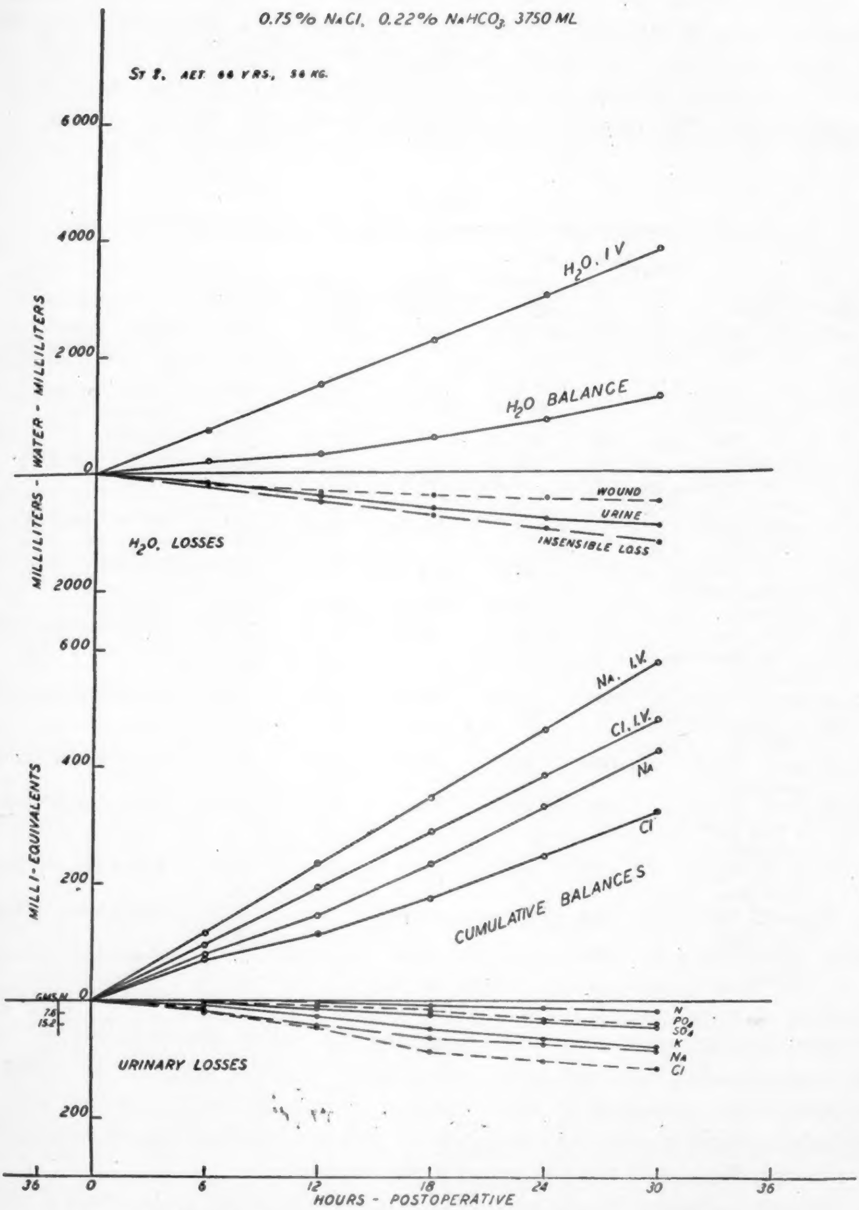

Fig. 2.—Cumulative balances of water, sodium and chloride, and losses of water and urinary constituents of patient St., f., No. 563,726, who received 3750 Ml. of 0.75% NaCl plus 0.22% $NaHCO_3$ during 30 hours following combined abdominoperineal resection. Operation was performed under continuous procaine spinal anesthesia. There was marked hypotension during the operation, and the postoperative course was characterized by tachycardia and hypotension. The patient received a transfusion of 500 Ml. of blood at the end of the infusion period.

potassium" excreted by all the patients studied. Judging from the losses of Levanzin, about 40 mEq. of the potassium are due to starvation. The remainder may be attributed in part to tissue trauma, in part to the infusion of 34 Gm. of salt. As shown by Gamble,[22] the addition of extracellular electrolytes causes a large transfer of intracellular water to the extracellular compartment. The removal of intracellular electrolyte follows in order to

TABLE II

URINARY EXCRETION OF POTASSIUM, SODIUM AND CHLORIDE IN 24 HOURS POSTOPERATIVELY

Patient—Infusion	Urine Volume Ml.	Potassium mEq./l.	Sodium mEq./l.	Chloride mEq./l.	Ratio Na:Cl	Comment
Br ♂ 0.9% NaCl (3000 Ml.)	886	97.9	105.2	165.0	0.64	Miles' regimen not used. Wound loss: 630 Gm.
Ke ♂ 0.9% NaCl (3000 Ml.)	579	92.5	94.6	147.3	0.64	Infusion in one dose postoperatively
Br ♀ 0.9% NaCl (3000 Ml.)	891	117.4	93.8	135.2	0.69	First day
	831	80.1	25.1	53.9	0.47	Infection, 2nd day
Mo ♀ 0.9% NaCl (3000 Ml.)	944	54.8	168.0	216.1	0.78	
Li ♀ 0.9% NaCl (3000 Ml.)	990	46.3	187.6	232.6	0.81	500 Ml. blood postoperatively
Fe ♂ 0.75% NaCl + 0.22% $NaHCO_3$	1230	116.7	168.0	154.3	1.09	First day
(3000 Ml.)	1380	81.0	154.8	150.5	1.03	Second day
St ♀ 0.75% NaCl + 0.22% $NaHCO_3$ (3000 Ml.)	735	87.0	98.0	126.0	0.78	Wound loss: 395 Gm.
Vo ♂ 0.75% NaCl + 0.22% $NaHCO_3$ (3000 Ml.)	1070	129.2	120.7	81.3	1.48	Wound loss: 610 Gm.
Mo ♂ 0.6% NaCl (4500 Ml.)	1186	100.6	91.0	124.8	0.73	Wound loss: 665 Gm.
Hu ♂ 0.38% NaCl + 0.11% $NaHCO_3$ (6000 Ml.)	895	101.3	138.3	112.1	1.23	Wound loss: 400 Gm.
Le ♂ 0.45% NaCl (6000 Ml.)	1170	129.5	121.2	141.5	0.86	Wound loss: 1375 Gm.
Re ♂ 0.45% NaCl (6000 Ml.)	491	106.5	15.2	52.5	0.29	Wound loss: 1826 Gm.
Sm ♀ 0.45% NaCl (4800 Ml.)	1321	72.1	95.4	102.1	0.93	Wound loss: 991 Gm.

preserve normal ionic concentration during the period of stress created by the load of salt. However, this explanation is scarcely adequate to account for the excess potassium in those cases receiving the large infusions which provided sufficient water. From Table II, it is evident that loss of potassium can be correlated with the volume of urine excreted.

Figure 1, Patient Br, ♂, 3,750 Ml. of 0.9 per cent NaCl, describes a very small balance of water at the end of 30 hours, a large load of sodium and chloride, a moderate loss of fluid from the posterior wound. The urinary excretion of chloride exceeded that of sodium.

Figure 2, Patient St, ♀, 3,750 Ml. of 0.75 per cent NaCl and 0.22 per cent $NaHCO_3$, indicates a greater load of water and a greater load of sodium and chloride, but the actual retention concentrations are less than those of

Figure 1. More chloride than sodium is lost in the urine, but the difference is small.

Figure 3, Patient Mo, ♂, 5,625 Ml. of 0.6 per cent NaCl, describes a positive load of water, a marked diuresis after 24 hours (a fact established by Bayliss and Fee[23] for 0.6 per cent NaCl in dogs), a minimal loss from wounds, large urinary "excess potassium" and a greater excretion of urinary

TABLE III

THIRTY-HOUR LOADS OF SODIUM, CHLORIDE AND WATER

Patient—Infusion		In-put mEq. or Ml.	Oxidative Water Ml.	Excretion Urine mEq. or Ml.	 Wound mEq. or Ml.	Insensible Loss Ml.	Load mEq. or Ml.
Br 0.9% NaCl	Na	577.5		119.6	93.0		364.9
	Cl	577.5		205.0	72.6		299.9
	H_2O	3750	161	1174	694	1470	573
St 0.75% NaCl+	Na	579.5		85.8	65.5		428.2
0.22% $NaHCO_3$	Cl	481.0		118.1	49.5		323.4
	H_2O	3750	94	885	450	1180	1329
Vo 0.75% NaCl+	Na	579.5		158.0	110.6		310.9
0.22% $NaHCO_3$	Cl	481.0		112.6	86.9		281.5
	H_2O	3750	218	1320	790	1640	218
Mo 0.6% NaCl	Na	577.5		189.0	120.2		268.3
	Cl	577.5		239.6	92.3		245.6
	H_2O	5625	200	2416	855	1470	1084
Hu 0.38% NaCl+	Na	577.5		263.5	119.7		194.7
0.11% $NaHCO_3$	Cl	577.5		212.8	90.5		177.7
	H_2O	7500	204	1495	905	1470	3834
Le 0.45% NaCl	Na	577.5		227.3	201.6		148.6
	Cl	577.5		264.5	152.9		150.1
	H_2O	7500	194	2130	1390	1510	2664
Sm 0.45% NaCl	Na	462.0		200.3	187.4		73.8
	Cl	462.0		225.6	142.2		85.5
	H_2O	6000	216	2044	1293	1010	1869
Re 0.45% NaCl	Na	577.5		10.9	375.8		190.8
	Cl	577.5		25.9	285.1		266.4
	H_2O	7500	79	506	2592	1470	3011

chloride. At the end of 48 hours, the patient had received 200 Ml. of orange juice and 250 Ml. of water, the sodium load was reduced to 164 mEq., the chloride load to 116 mEq. and the water load to —38 Ml.

Figures 4 and 5 are presented to contrast the paths of excretion of large volumes of fluid. Both patients received 7,500 Ml. of 0.45 per cent NaCl. Patient Le, ♂, Figure 4, showed a large load of water, a marked increase in the loss from wounds, a diuresis after 24 hours, a greater urinary excretion of chloride than of sodium, and the smallest load of NaCl of the patients charted. Patient Re, ♂, Figure 5, was hypotensive after the operation. The volume of urine excreted was very small, the quantity of sodium and chloride negligible. Such massive losses of fluid from the posterior wound occurred that the final loads of sodium, chloride and water were not unlike those of Figure 4.

Figure 6, Patient Hu, ♂, 7,500 Ml. of 0.375 per cent NaCl and 0.11 per cent $NaHCO_3$, shows the largest water retention of the group and a pronounced urinary loss of both sodium and chloride, with sodium predominating.

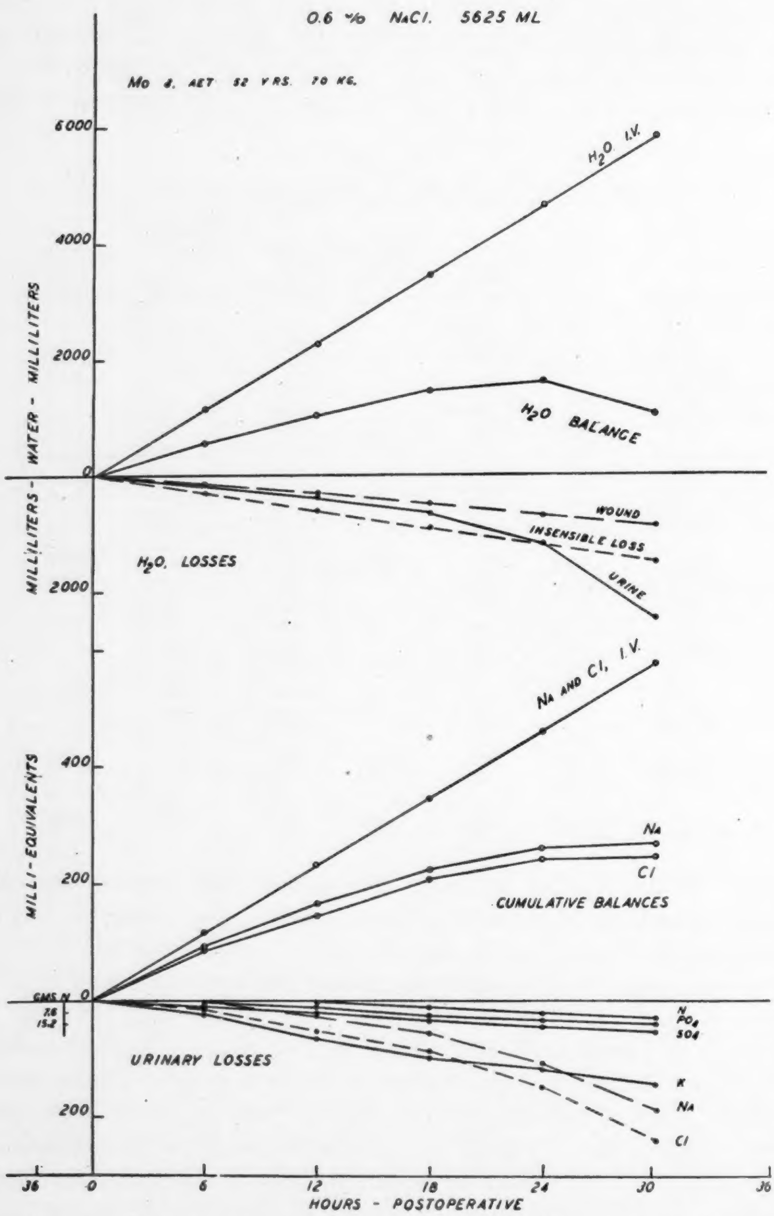

FIG. 3.—Cumulative balances of water, sodium and chloride, and losses of water and urinary constituents of patient Mo., m., No. 568,132, who received 5625 Ml. of 0.6% NaCl during 30 hours following combined abdominoperineal resection. Operation was performed under continuous procaine spinal anesthesia. Hypotension did not develop, and the postoperative condition of the patient was excellent.

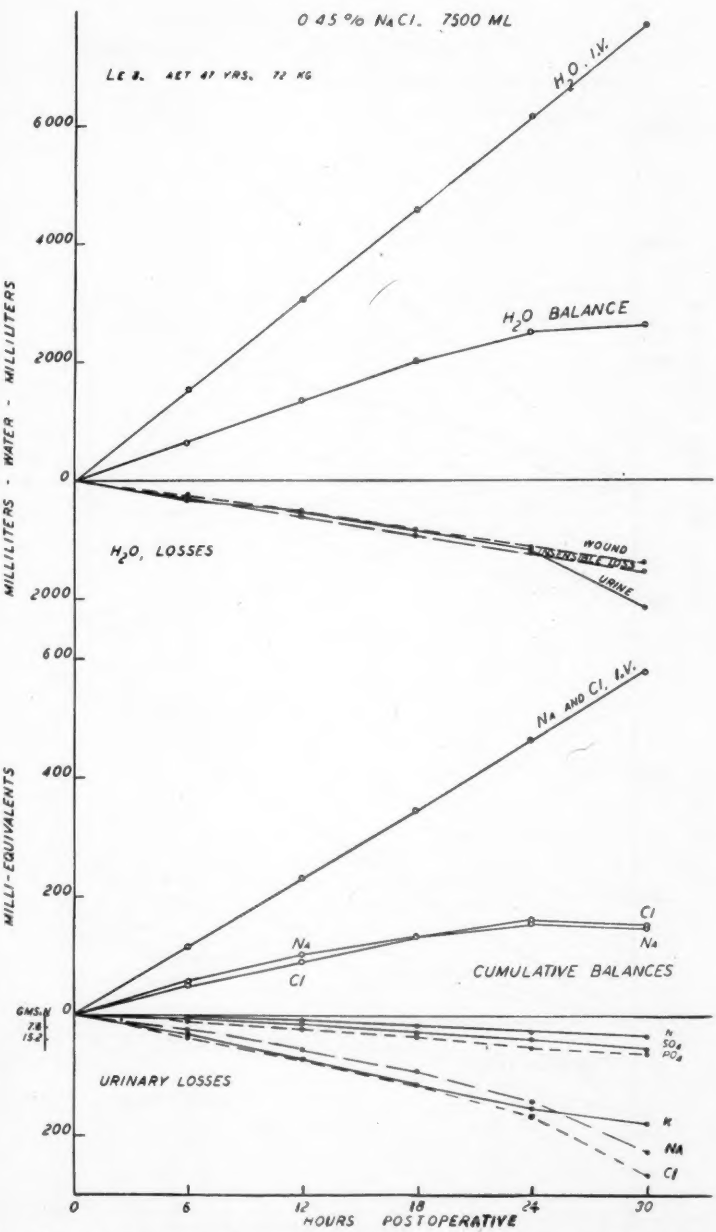

FIG. 4.—Cumulative balances of water, sodium and chloride, and fluid and urinary losses of patient Le., m., No. 564,071, who received 7500 Ml. of 0.45% NaCl during 30 hours following combined abdominoperineal resection. The operation was performed under nupercaine spinal anesthesia. Operative and postoperative courses were uneventful.

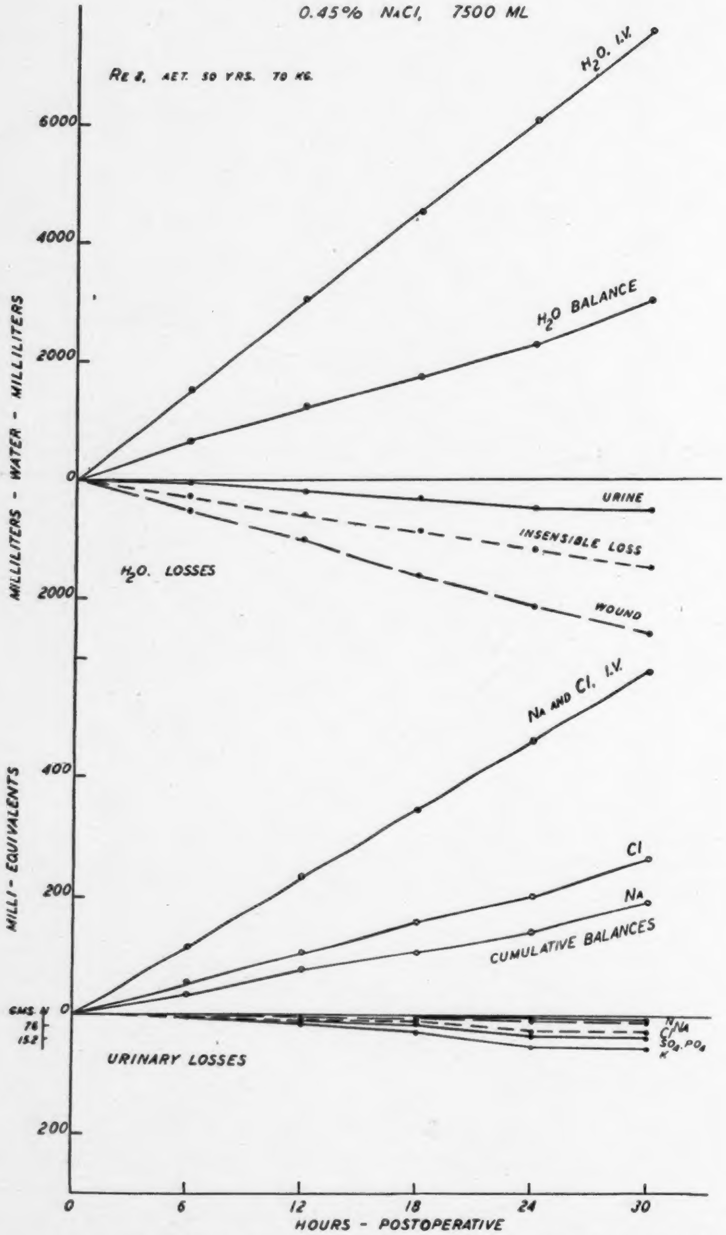

FIG. 5.—Cumulative balances of water and sodium and chloride, and fluid and urinary losses of patient Re., m., No. 562,197, who received 7500 Ml. of 0.45% NaCl during 30 hours following operation. An uneventful operation was followed by a prolonged period (12 hours) of hypotension, very low pulse pressure, tachycardia and oliguria. The entire postoperative course was characterized by a very marked transudation from the posterior wound.

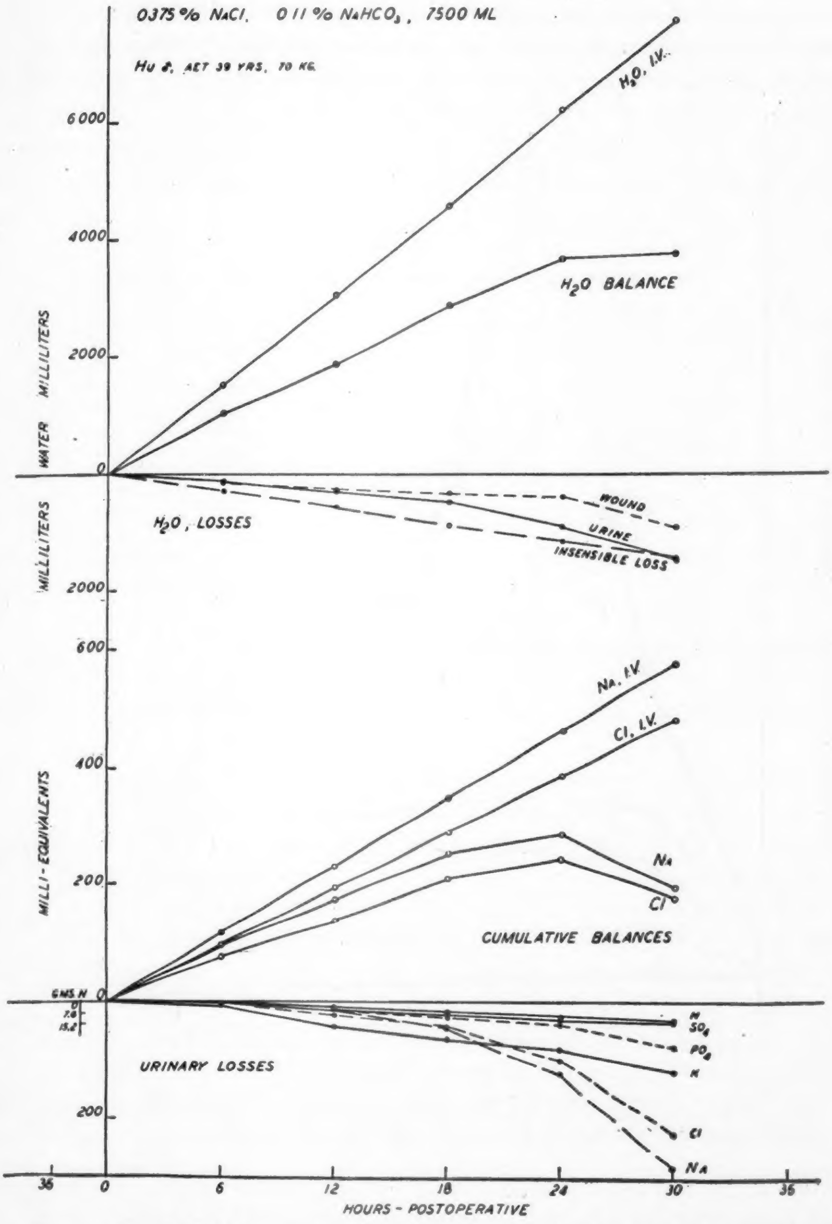

FIG. 6.—Cumulative balances of water and sodium and chloride, and fluid and urinary losses of patient Hu., m., No. 568,596, who received 7500 Ml. of 0.38% NaCl plus 0.11% $NaHCO_3$ during 30 hours following operation. Mild hypotension developed during the operation, 60/40. The postoperative course was satisfactory.

The loss from wounds is small, in contrast to the patients receiving 0.45 per cent NaCl. At the end of 48 hours, the patient had reduced the sodium load to —10 mEq., the chloride load to zero, and the water load to 2,770 Ml.

Table IV divides the patients into a so-called physiologic salt group (physiologic sodium or physiologic chloride) and an hypotonic group. The loads at 30 hours are computed as per cent of in-put. Basic differences are

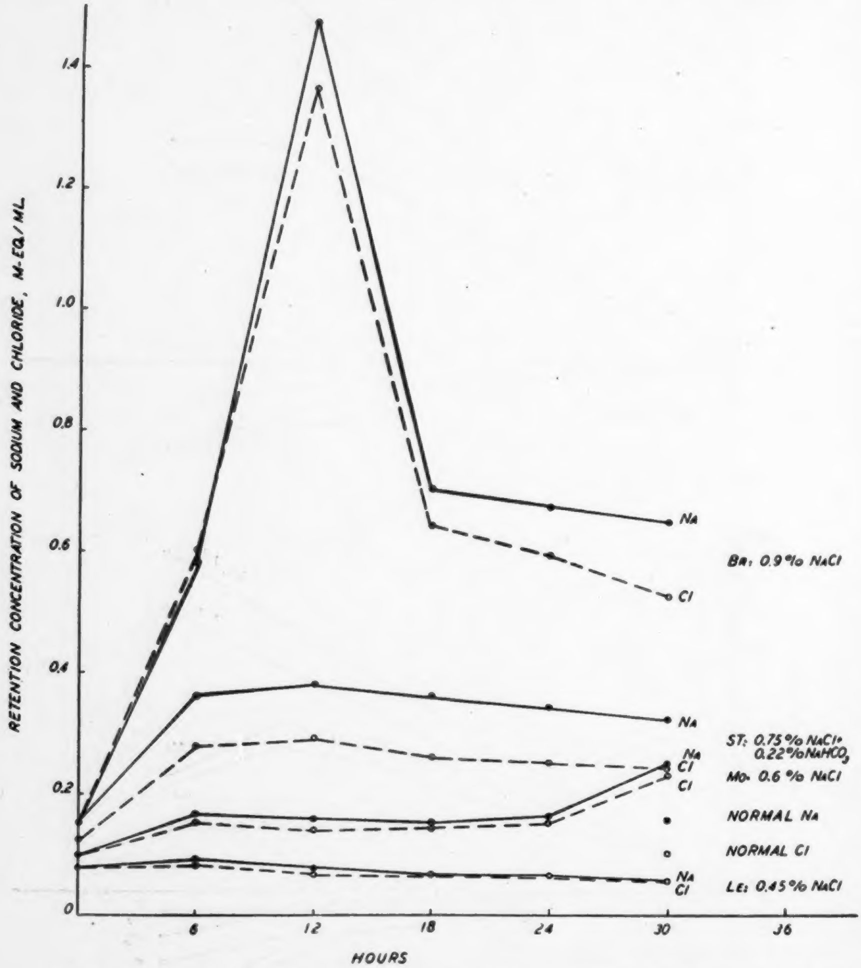

FIG. 7.—Calculated retention concentrations of sodium and chloride, mEq. per Ml. of four patients receiving isotonic salt solution, adjusted isotonic salt solution, isotonic chloride and hypotonic salt solutions, plotted against time after operation in hours.

found in the marked retention of salt with respect to water in the physiologic salt group, and the retention of water over salt in the hypotonic group.

Figure 7 describes the same findings graphically by plotting retention concentrations of sodium and chloride against time in hours. The graph reveals no tendency of the human kidney to convert the volume of infused fluid to a retained concentration of 0.102 mEq. per Ml. as found by Wolf[24] for the

dog, and again emphasizes the inability of the human kidney to concentrate urine so well as the dog.

Discussion.—Contrary to animal experiments, surgical patients cannot be considered to have a zero salt and water load at the start of the operation. Varying degrees of hydration and salt balance must exist, especially if the patient has been prepared for operation by Miles' regimen, as in the present series. However, the retention concentrations of Patient Br indicate strongly that there is little need for 27 Gm. of salt. The average loss of blood at these operations indicates a need of about 2 Gm. of salt. The urine, at best,

Table IV

THIRTY-FOUR LOADS OF SODIUM, CHLORIDE AND WATER, PER CENT OF IN-PUT

Patient	Infusion	Sodium Per Cent	Chloride Per Cent	Water Per Cent
Br ♂	0.9% NaCl	63	52	15
St ♀	0.75% NaCl+0.22% $NaHCO_3$	74	67	35
Vo ♂	0.75% NaCl+0.22% $NaHCO_3$	27	23	5
Mo ♂	0.6% NaCl	46	43	19
Average, isotonic solutions		53	46	19
Hu ♂	0.38% NaCl+0.11% $NaHCO_3$	34	37	50
Le ♂	0.45% NaCl	26	26	34
Sm* ♀	0.45% NaCl	16	19	30
Re ♂	0.45% NaCl	33	46	40
Average, hypotonic solutions		27	32	39

* Sm received 80% of the infusion.

excretes 7 Gm. of the excess; the losses from the anterior and posterior wounds accommodate an additional 5 Gm. There remains an excess of 13 Gm. of salt which serves only to embarrass a water balance already strained by the exigencies of operation and sequelae.

The hypothesis that the excretion rate of a substance is ordinarily proportional to load is untenable in the case of these surgical patients. The normal kidney is able to concentrate chloride taken orally to the extent of 0.29 to 0.33 mEq. per Ml.[25] In spite of heavy salt loads, no patient approximated this value. As a result of increasing salt load and increasing hypertonicity of the extracellular compartment, osmotic relationships can only be maintained by a shift of water from intracellular to extracellular space. Loads of salt created by the isotonic solutions require a transfer of approximately two liters of intracellular water within 30 hours after operation. The edema, a symptom of postoperative salt intolerance, may result not so much from the retention of water with salt as from the shifting of water from the intracellular to the extracellular space. It is unknown how much dehydration the cells can undergo before function breaks down and ceases. The brain cells are especially sensitive to change and the disorientation so often seen in cases of salt intolerance may be a symptom of this fluid shift.

SUMMARY AND CONCLUSIONS

Salt solutions of various composition and tonicity were administered to men and women undergoing major surgical operations. The rates of excre-

tion of the urinary constituents were determined. Losses of sodium, chloride and water from the posterior and anterior wounds were estimated by use of weighed dressings. Cumulative balances of water, sodium and chloride were calculated.

1. The injection of "isotonic sodium chloride" solutions was attended by an average retention of 53 per cent of the sodium, 46 per cent of the chloride, and 19 per cent of the water 30 hours after the operation. Such retentions of salt indicate a withdrawal of approximately two liters of fluid from the intracellular compartment in order to maintain isotonicity.

2. The infusion of hypotonic solutions resulted in the average retention of 27 per cent of the sodium, 32 per cent of the chloride, and 39 per cent of the water during the same postoperative period. Extra water is thereby provided for excretory function of skin and lungs, and the intracellular compartment is not involved.

3. The human kidney, under the conditions of these experiments, does not elect to guard a "physiologic saline" solution.

4. If intravenous infusion is indicated in the postoperative care of the surgical patient, hypotonic solutions, 0.45 per cent NaCl, or better, 0.38 per cent NaCl plus 0.11 per cent $NaHCO_3$, should replace the "isotonic" solutions commonly in use.

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METABOLIC ALTERATIONS FOLLOWING THERMAL BURNS

V. THE USE OF WHOLE BLOOD AND AN ELECTROLYTE SOLUTION IN THE TREATMENT OF BURNED PATIENTS*

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In recent years there has been some controversy concerning the best means of combating shock in severely burned patients. Although there are a number of methods which apparently suffice to tide patients over the critical 48-hour period following an injury, their influence on the subsequent course of the patients is not well understood. In 1944, Moyer, Coller, Iob, Vaughan and Marty[1] employed various forms of therapy in the treatment of shock caused by a burn involving 80 per cent of the body of anesthetized dogs. They pointed out that the dogs treated with whole blood intravenously and a sodium chloride-bicarbonate solution orally survived longer than dogs who were treated with plasma. Because their experiments were limited to the early shock period it seemed desirable to evaluate the effect of these various forms of treatment in less severely burned animals and to note the changes which occurred during the convalescent period. The results of such a study were reported in a previous publication.[2] They tended to show that the anemia which occurred during the convalescent period could be prevented if whole blood was given early.

It is the purpose of this communication to compare a group of patients who were treated with plasma with another group who received whole blood intravenously and an electrolyte solution orally. The changes observed in various blood constituents and in the elimination of water and salt will be briefly discussed.

METHODS

The patients who are included in this report were studied after being admitted to the Metabolic Ward. The organization of the Metabolic Ward and the facilities have been previously described.[3] The methods of determining the cell volume (hematocrit), plasma protein and albumin concentration have been previously stated.[4] The plasma chloride concentration was determined by the method of Van Slyke as modified by Wilson and

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Ball,[5] the plasma CO_2 content by the method of Van Slyke and Neill,[6] the blood urea was done as described by Van Slyke and Cullen,[7] and the non-protein nitrogen concentration determined by a modification of the method of Gentzkow.[8] Sodium and potassium were determined on the aqueous solution of the ash, digestion and ashing carried out according to the method of Cullen and Wilkins,[9] with the ashing temperature maintained at 450°-500° C. Sodium was determined by the method of Butler and Tuthill's[10] modification of Barber and Koltoff, and potassium by the method of Shohl and Bennett as modified by Salit.[11] The blood was drawn with a minimum amount of stasis, employing greased syringes, and clotting was prevented by the use of heparin. All samples for CO_2 content were drawn and separated under oil and again heparin was employed as the anti-coagulant. Blister fluid taken from A. T. was aspirated by inserting a sterile needle through normal tissue into the bottom of the blister. The fluid was removed with an oiled syringe containing heparin. A small number of red blood cells which were present in the samples were removed by centrifugation before the clear fluid was analyzed.

The electrolyte solution* contained the following amount of salts per liter of distilled water: 6.10 Gm. sodium chloride; 0.20 Gm. calcium chloride; 0.20 Gm. potassium chloride; 0.07 Gm. sodium phosphate (monobasic); 0.05 Gm. magnesium chloride; 2.38 Gm. sodium bicarbonate; and 2.0 Gm. dextrose. The approximate amount of this solution given over the first 48 hours was equal to 15 per cent of the patient's body weight. The quantities of blood and plasma employed were prescribed according to the severity of shock, depth and the surface area involved, and the size of the patient.

RESULTS

For the convenience of presentation and discussion, the patients are divided into mildly burned (5-15 per cent of the surface area); moderately burned (15-35 per cent); and severely burned patients (35-100 per cent).

In Figure 1 the changes in the hematocrit, the extent and type of burn, and the therapy in six mildly burned patients are presented. I. S., Case 2, and D. C., Case 3, received no intravenous therapy, while varying amounts of plasma were given to the remaining cases. It is interesting to note that in all of these patients, with the possible exception of D. C., the hematocrit fell to levels below normal. Hemoconcentration was not evident except in the case of D. C. The plasma protein concentration in these six patients remained above 6.0 Gm. per 100 cc. but in many instances the initial plasma albumin concentration of 4.0 to 4.5 Gm. per 100 cc. subsequently fell to around 3 Gm. per 100 cc. The plasma chloride concentration and blood urea levels were within the normal range while in most cases the hemoglobin levels and red blood cell counts paralleled the change in the hematocrit (on several occasions the hemoglobin showed a slightly more marked fall than did the

* Kindly furnished in concentrated form in sterile ampoules by Sharp & Dohme, Philadelphia, Pa.

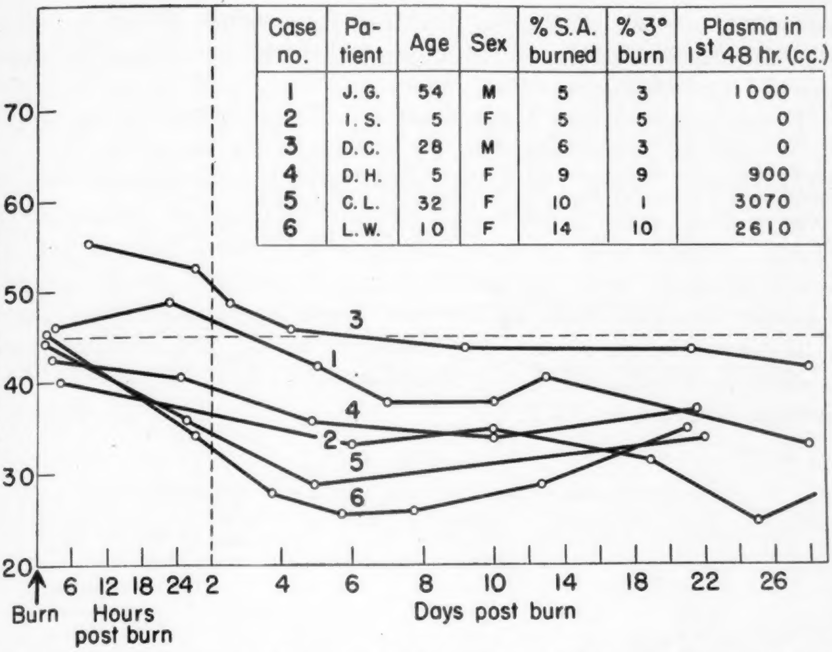
hematocrit). The burn was caused by clothing catching on fire in all instances except that of C. L., Case 5, who was burned during an explosion (flash burn).

Figure 2 shows the alterations in the hematocrit in five moderately burned patients who were treated with plasma intravenously. The hematocrit again showed a significant decrease below the normal during the convalescent phase and the hemoglobin showed a decrease as great or slightly greater than the hematocrit. Hemoconcentration was evident only in Cases 8 and 11 (Fig. 2). The plasma protein concentration, as a rule, was above 6 Gm. per 100 cc., but in H. S. and M. M. it fell to 5.5 Gm. during the first week post-burn and then rose slowly towards the normal. In L. S. it decreased gradually to 4.5 Gm. per 100 cc. on the third day post-burn. The plasma albumin concentration decreased in all of the patients and in M. M., H. S. and L. S. it reached 2.7, 2.8 and 2.5 Gm. per 100 cc., respectively, when the total protein concentrations were at their lowest level. The blood urea concentrations remained normal in all of these cases with the exception of L. S. This five-year-old patient was burned when her clothing caught fire and, although therapy was instituted promptly, she became anuric shortly thereafter and remained so until death on the fourth hospital day. She had a previous history of nephritis and it was felt that the renal shut-down was in a great part due to the preëxisting disease. Various solutions were given intravenously to this patient (10 per cent glucose, one-sixth molar sodium lactate, and blood) in an attempt to increase the urinary output of the kidneys, but without avail.

Figure 3 shows the alterations in the hematocrit that occurred in four mildly and moderately burned patients treated with an electrolyte solution orally. In three patients whole blood was also administered. In J. J., Case 14, blood was not given initially because the patient was seven months pregnant and was *Rh-negative*. About 30 hours after being burned a premature baby was delivered, but in spite of this and a fairly severe burn, the patient's convalescence was quite uneventful. She did receive several subsequent transfusions of *Rh-negative* blood.

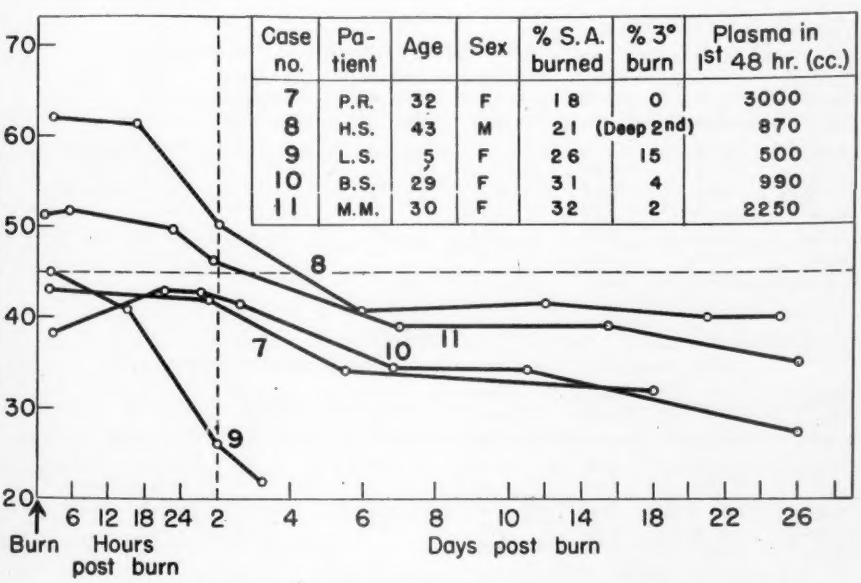
In these cases the hematocrit showed no appreciable decrease, the blood urea concentration remained within a normal range throughout, and the plasma protein and albumin concentrations paralleled quite closely the changes seen in the patients treated with plasma. In the first two patients, normal values were encountered throughout, but in J. J. and B. T. the plasma protein concentration dropped to 5.03 and 5.12 Gm., respectively, but rose gradually to 6.00 Gm. per 100 cc., and remained at that level, or above, for the remainder of their hospital stay. The plasma albumin concentrations in these two patients, likewise, showed a fall to around 2.3 Gm. per 100 cc. when the plasma protein concentration was low.

In most of the patients presented in the first three figures, the plasma albumin concentration fell after the burn, reaching its lowest value somewhere between three to ten days. It later increased, but rarely did it reach the



Case no.	Patient	Age	Sex	% S.A. burned	% 3° burn	Plasma in 1st 48 hr. (cc.)
1	J. G.	54	M	5	3	1000
2	I. S.	5	F	5	5	0
3	D. C.	28	M	6	3	0
4	D. H.	5	F	9	9	900
5	C. L.	32	F	10	1	3070
6	L. W.	10	F	14	10	2610

FIG. 1.—Hematocrit alterations in mildly burned patients receiving plasma or no intravenous therapy.



Case no.	Patient	Age	Sex	% S.A. burned	% 3° burn	Plasma in 1st 48 hr. (cc.)
7	P.R.	32	F	18	0	3000
8	H.S.	43	M	21	(Deep 2nd)	870
9	L.S.	5	F	26	15	500
10	B.S.	29	F	31	4	990
11	M.M.	30	F	32	2	2250

FIG. 2.—Hematocrit alterations in moderately burned patients treated with plasma.

value noted on admission, even though the plasma protein concentration had returned to normal. Thus, the plasma globulin values showed an increase while the plasma albumin concentration was lowered, and this condition often persisted for weeks.

Hemoconcentration of a significant degree was evident in only three of the 15 patients, as determined by an increase in the hematocrit.

In Tables I, II and III, the treatment given and the chemical alterations noted in three severely fire-burned patients who received plasma are presented. It can be seen from the findings in these three patients that at the time of death the hematocrit and the plasma protein and albumin concentrations had decreased below the normal. While hemoconcentration occurred, it did not parallel the severity of the patients' burns. The nitrogenous waste products were well cleared from the blood in Cases C. W. and F. H., and partially removed in A. T.

TABLE I

C. W. 42-YR.-OLD. ♂ BURNED BY FIRE. TOTAL BURN 32%; 3°-BURN 25%

Time Post-burn	Hematocrit	Plasma Protein Gm./100 Cc.	Plasma Albumin Gm./100 Cc.	Blood Urea Mg./100 Cc.	Plasma Chloride mEq./liter	Treatment
3.5 hrs.	49.6	6.43	4.05			900 cc. plasma
10 hrs.	51.5	6.40			102.3	885 cc. plasma
16.5 hrs.	60.0					600 cc. plasma
34 hrs.	58.5	5.80	3.10	35		1060 cc. plasma
2.25 days	51.5	5.30	2.90			1165 cc. plasma
5 days	44.0	5.30		30		——
7 days	38.2	4.60	2.40	28	98.0	4610 cc. total plasma given

TABLE II

A. T. 48-YR.-OLD. ♂ BURNED BY FIRE. TOTAL BURNS 45%; 3°-BURN 35%

Time Post-burn	Hematocrit	Plasma Protein Gm./100 Cc.	Plasma Albumin Gm./100 Cc.	Nonprotein Nitrogen Mg./100 Cc.	Plasma Chloride mEq./liter	Treatment
7 hrs.	66.0					1500 cc. plasma
1 day	70.4	6.89			98.8	3035 cc. plasma
2.33 days	51.6	5.27	3.21	82	101.0	3000 cc. plasma
4 days	51.6	5.70	2.79	93	93.6	——
6 days	41.8	5.35	2.79	82	94.4	7535 cc. total plasma given
9 days	37.9	5.24	2.42	90	103.8	
12 days	36.2	5.56	2.44	70	102.8	

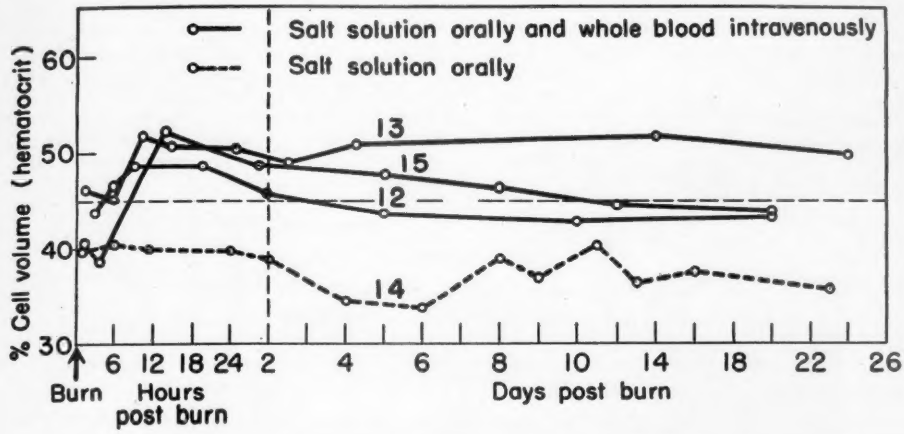
TABLE III

F. H. 17-YR.-OLD. ♀ BURNED BY FIRE. TOTAL BURN 64%; 3°-BURN 32%

Time Post-burn	Hematocrit	Plasma Protein Gm./100 Cc.	Plasma Albumin Gm./100 Cc.	Blood Urea Mg./100 Cc.	Plasma Chloride mEq./liter	Treatment
2 hrs.	59.5	6.60	4.19	37		
1 day	43.4	5.06	3.00			4240 cc. plasma
2 days	36.0	6.01	3.10	47	106.0	1500 cc. plasma
3 days	39.4	5.30	2.90	27		——
5 days	34.2	5.19	2.75		96.2	5740 cc. total plasma given

In Tables IV, V and VI, the alterations in the blood chemistry which occurred in three patients burned by fire and treated with whole blood intravenously and an electrolyte solution orally are presented. The decrease in

the hematocrit did not occur until after a month although whole blood was only given during the first 24 hours after the burn. There was a fairly marked decrease in the total plasma protein and albumin concentration in all three cases, but this in no way seemed to interfere with the recovery of patients M. D. and W. L.



Case no.	Patient	Age	Sex	% S.A. burned	% 3° burn	Solution given in first 48 hr. (cc.)	
						Electrolyte solu. orally	Blood I. V.
12	J. J. J.	46	M	12	0	9000	450
13	T. A.	14	M	14	1	8000	800
14	J. J.	25	F	25	18	10000	0
15	B. T.	3	F	35	0	3000	500

FIG. 3.—Hematocrit alterations in mildly and moderately burned patients treated with an electrolyte solution orally.

Edema was present in these three patients but was decreasing by the third to fourth day post-burn and was not clinically evident after the sixth day. The patients (Tables I, II and III) who were treated with plasma showed just as marked an accumulation of fluid which persisted somewhat longer.

Figures 4, 5, 6, 7 and 8 show the total fluid intake and urinary output in five of the aforementioned cases. It can be observed that in the mildly and moderately burned cases (J. J. J., W. L., and M. D.) a much higher per cent of the total fluid ingested was eliminated than in the more severely burned patients (A. T. and R. J.). The findings in these five cases were quite comparable to the other patients in whom similar studies were made. Thus, the records of the patients studied show that following a burn, larger amounts of fluid are consumed than would be normally. In those who ultimately recover the elimination of the excess fluid is readily accom-

TABLE IV

W. L. 11-YR.-OLD. ♂ BURNED BY FIRE. TOTAL BURN 28%; 3°-BURN 12%

Time Post-burn	Hemato-crit	Plasma Protein Gm./ 100 Cc.	Plasma Albumin Gm./ 100 Cc.	Nonprotein Nitrogen Mg./ 100 Cc.	Plasma Chloride mEq./ liter	Plasma CO_2 Content Vol. %	Plasma Amino-acid Mg./ 100 Cc.	Treatment
3.5 hrs.	57.0	6.18	4.03	30	97.6	47.5	3.78	500 cc. blood
17 hrs.	58.0	4.66	2.79	18	106.2		5.34	
23 hrs.	59.6							500 cc. blood (5000
29.5 hrs.	60.0							cc. of the electrolyte
39 hrs.	58.9	4.58	2.46		97.5		6.17	solution plus 1000 cc.
3 days	52.8						4.78	of water taken first
5 days	46.2	5.11	2.53	33	93.4	56.6	3.92	day, and 2000 cc. of
9 days	43.8	5.91	2.84	24	97.2	61.6		the electrolyte solu-
12 days	45.5				96.4	58.0	3.39	tion plus 1000 cc. of
18 days	41.2	6.66	3.29	37	104.1	59.3	3.65	water on second day)
30 days	31.7	6.16	3.44		104.2			

TABLE V

M. D. 26-YR.-OLD. ♀ BURNED BY FIRE. TOTAL BURN 32%; 3°-BURN 20%

Time Post-burn	Hemato-crit	Plasma Protein Gm./ 100 Cc.	Plasma Albumin Gm./ 100 Cc.	Nonprotein Nitrogen Mg./ 100 Cc.	Plasma Chloride mEq./ liter	Plasma CO_2 Content Vol. %	Sodium mEq./ liter	Treatment
1.5 hrs.	51.3	7.12	4.47	21	100.9			1520 cc. blood (6000
29 hrs.	59.2	5.29	3.24	26	100.9	49.0		cc. of electrolyte so-
2.25 days	57.2	5.47	3.41	18	99.1			lution during first 24
4 days	49.8	5.03	2.79	25	91.2	49.0		hrs., and 2000 cc. of
6 days	48.8	5.42	2.85	25	88.4	66.0		electrolyte solution
8 days	47.0	5.91	2.61	30	87.3	70.8		given on second day)
11 days	44.2	6.10	2.59	25	98.6	55:1	135:2	
14 days	45.9	6.76	2.89	29	94.3	56.0		
19 days	44.4	7.26	3.01	32	98.6	63.3	135.2	
22 days	41.3	7.18	3.14	31	93.6			
29 days	28.3		2.69	22	91:7	61.3		
42 days	34.4	6.63	3.07	26	100.1	62.0	139.2	
60 days	38.9	6.45	3.37	24	102.3			
74 days	40.7	6.55	3.51		99.0			
90 days	40.8	7.09	4.01	31	102.4			

TABLE VI

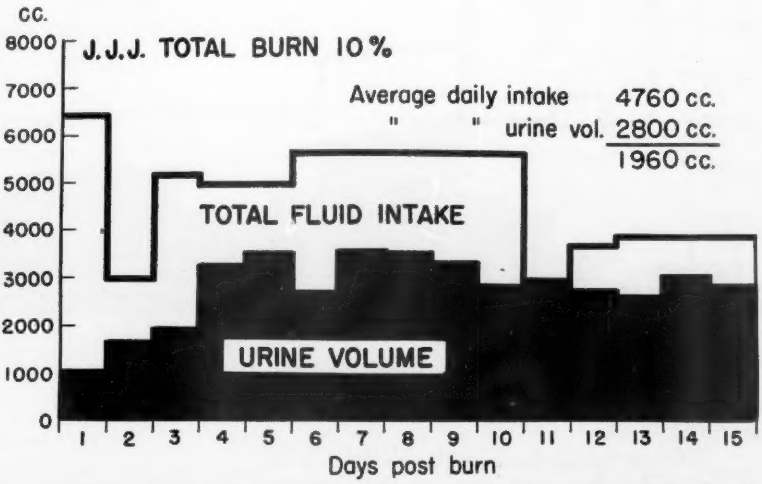
R. J. 42-YR.-OLD. ♂ BURNED BY FIRE. TOTAL BURN 47%; 3°-BURN 35%

Time Post-burn	Hemato-crit	Plasma Protein Gm./ 100 Cc.	Plasma Albumin Gm./ 100 Cc.	Nonprotein Nitrogen Mg./ 100 Cc.	Plasma Chloride mEq./ liter	Plasma CO_2 Content Vol. %	Plasma Amino-acid Mg./ 100 Cc.	Treatment
2.5 hrs.	59.8	6:99	4:77	26	101:2	47.7	5.90	470 cc. blood
8 hrs.	76.1				106.8	46.5	6.67	460 cc. blood
13 hrs.	75.6				105.9		6.89	250 cc. blood
27 hrs.	72.4	4:14		65	113.9	52.5	4.70	530 cc. blood
34 hrs.	63.6	3.99			117.6			
52 hrs.	62.1	4.40		52	112.5	63.2	5.07	
75 hrs.	54.3	4.28	2:12	64	112.2	55.6	4.61	350 cc. conc. plasma
81 hrs.	46.2	4.08			112.1			
84 hrs.	47.6							
4 days	47.0	4.72			111:2		5.53	550 cc. blood (Pt.
5 days	46.3	5.20	2.24	75	111.1		4.34	took 9000 cc. of the
7 days	43.5	5:52		158		40 6	6.50	electrolyte solution
								on the first day, and
								3000 cc. on the sec-
								ond day post-burn)

plished, but in the cases that die there seems to be an inability of the body to properly rid itself of water.

It is difficult to tell how much fluid was lost in the exudate from the burned area, as perspiration, and through the lungs, but it was felt that the loss through these sources was not greater in the severely burned patients than in those with moderate burns. In many instances, where an extensive

Fig. 4



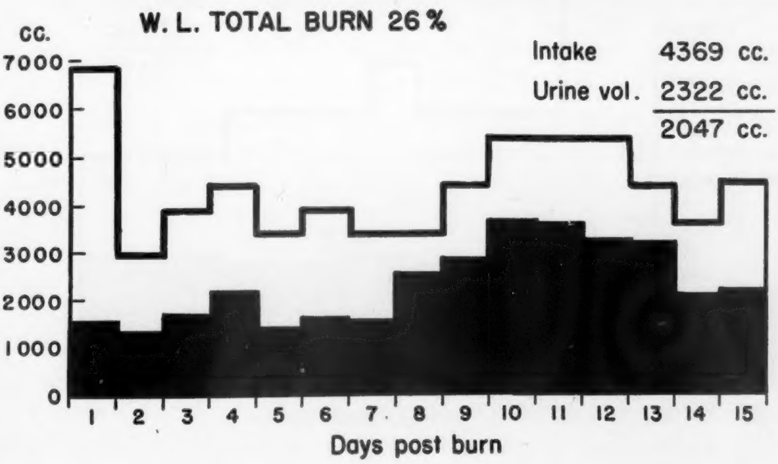


Fig. 5

Fig. 4.—Total fluid intake and urine excretion in a mildly burned patient (no third-degree burn).

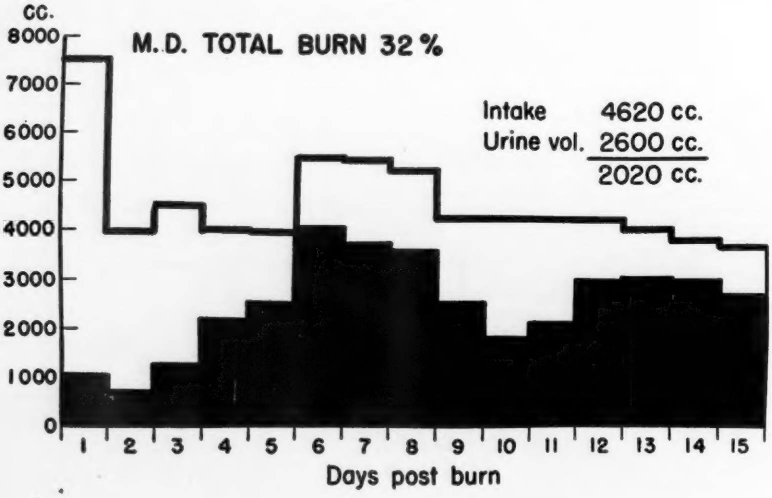
Fig. 5.—Total fluid intake and urine excretion in a moderately burned patient (12% of surface area involved by third-degree burn).

third-degree burn was present the skin was of a leathery consistency and very little fluid could penetrate through. Thus, while it is not possible to tell for certain it seems likely that patients A. T. and R. J. retained much greater amounts of fluid than did the other cases that survived.

The analysis of the blister fluid obtained from A. T., and the time post-burn that each specimen was removed, are shown in Table VII.

The daily loss of electrolytes from the exudate was measured in ten of

FIG. 6



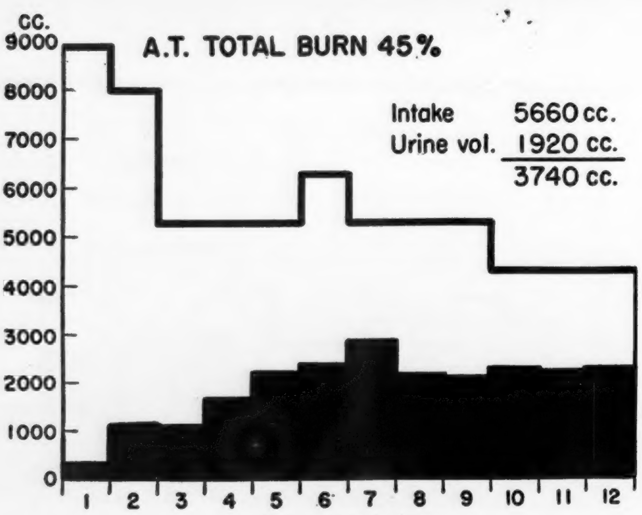


FIG. 7

FIG. 6.—Total fluid intake and urine excretion in a moderately burned patient (20% of surface area involved by third-degree burn).

FIG. 7.—Total fluid intake and urine excretion in a severely burned patient (35% of surface area involved by third-degree burn).

the patients as well as the intake and urinary output of sodium, potassium and chloride and will be reported in a later publication. It seems evident from this work, however, that the external loss (urine, feces and exudate) of the various electrolytes studied parallels somewhat the elimination of

water and is a great deal less in the severely burned patient than in the mildly or moderately burned cases.

Discussion.—From the work presented and previous experimental work[2] it would seem that the giving of whole blood early prevents or alleviates the anemia which occurs so commonly during convalescence from a burn. The various causes of the anemia seen during the convalescent period have been previously discussed,[2] and it was brought out that a state of overhydration may, in many instances, contribute to a decrease in the hematocrit, and also in the plasma protein concentration. It is felt that Case 9 (L. S., Fig. 2) is an example of this. This patient was given large amounts of fluid which she was unable to excrete and since a relatively small amount of blood and plasma was administered, the precipitous fall noted in her hematocrit and in the plasma protein concentration was for the most part due to a dilution of these constituents rather than to a diminution in the actual amount of blood cells and protein.

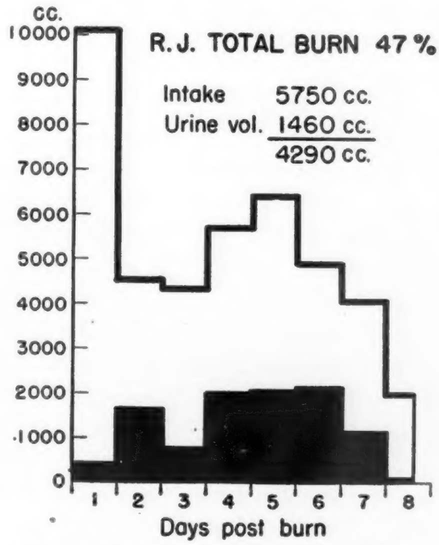


Fig. 8.—Total fluid intake and urine excretion in a severely burned patient (35% of surface area involved by third-degree burn).

From the experimental work of Rosenthal,[12] Allen,[13] and Warren, Merrill and Stead,[14] and the clinical experience of Fox,[15] it appears that solutions containing sodium salts are beneficial in the treatment of shock. One of our cases (J. J., Case 14) who was quite severely burned was treated only with an electrolyte solution by mouth during the period of shock. The remaining cases received whole blood intravenously and an electrolyte solution orally, as advocated by Moyer, Coller, Iob, Vaughan and Marty.[1]

Although this series of cases is much too small to permit definite conclusions regarding the effect of various types of therapy on the mortality rate, it does appear that the judicious use of whole blood and an electrolyte solution is an effective means of combating shock in burned patients.

Patients with a severe or moderate burn accumulate large amounts of fluid in the traumatized area regardless of the type of therapy employed to combat shock, but it has seemed that the edema diminishes more rapidly when salt solutions were used than when large amounts of plasma were administered. When plasma is given much additional protein is provided but there is no evidence to show that the plasma protein in itself is of more than temporary value in these burned patients. In some of our patients who received large amounts of plasma, low protein values were encountered and in three of the severely burned patients these low values persisted until death. Low plasma

protein levels were also encountered in the patients treated with smaller quantities of citrated whole blood and an electrolyte solution orally, but these were usually seen when the edema was decreasing and subsequently the protein levels rose toward normal. There is no reason to suspect that the giving of whole blood would be more beneficial than plasma in the prevention of hypoproteinemia, but the evidence does indicate that the giving of plasma does not prevent the decrease in the plasma protein concentration.

It has been generally believed that by increasing the blood osmotic pressure by the addition of plasma proteins, especially albumin, shock could be prevented or corrected. In peripheral circulatory collapse resulting from a burn, however, the albumin is rapidly lost, and, thus, gives only a transient beneficial effect. The analysis of the blister fluid taken from A. T. (Table VII) shows that the greatest part of the protein present in such fluid is albumin. These results are somewhat comparable to those of McIver,[16] and of Harkins,[17] except that the figures for protein and nonprotein nitrogen in this case are higher (probably due to a more severe burn). McIver[16] reported that the concentration of sugar (87 mg. per 100 cc.) paralleled that found in the blood, and stated that the calcium concentration (15.3 mg. per 100 cc.) was slightly elevated. Previous studies on sodium and potassium concentrations have not, as far as we are aware, been reported. These values tend to show a slight increase in the first 27 hours above the normal plasma concentrations.

TABLE VII

ANALYSIS OF BLISTER FLUID (A. T., 48-YR.-OLD ♂ 45% OF BODY BURNED)

Time Post-burn	Total Protein Gm./100 Cc.	Albumin Gm./100 Cc.	Sodium mEq./liter	Potassium mEq./liter	Chloride mEq./liter	Nonprotein Nitrogen Mg./100 Cc.	CO_2 Vol. %
27 hrs.	5.51	3.76	150.0	6.05	101.6	74	
4 days	5.06	3.15			98.8	102	55.8
6 days	4.59	3.21	136.5	5.18	96.2	65	

Thus, most of the osmotic effect of albumin or of the entire plasma proteins is transient and when it is lost through the capillary walls into the injured area it must continue to exert an osmotic pull towards the injured tissues because of the protein which has then accumulated in that region.

Since much of the administered protein is lost in patients in shock resulting from a burn, it would seem as though the best treatment would be: (a) in giving a substance which exerted an osmotic effect and would not be lost; (b) in rendering the injured capillaries less permeable, if such were possible, or; (c) by increasing the tissue tension to a point where fluid would no longer tend to leave the capillaries in excessive amounts.

Danielli's[18] experiments tend to show that the edema which occurs when a salt solution alone is perfused through an animal's extremity can be slowed by adding blood cells (especially platelets) to the solution. The perfusion experiments of Zweifach[19] show that there is apparently a substance derived from the surface of fresh plasma which has a "coating effect" and, thus,

tends to decrease the capillary permeability. In view of this work it would appear as though whole blood or plasma would have some advantage over a salt solution alone, but apparently since capillary permeability cannot be quickly or completely restored to normal it would seem logical to give an electrolyte solution so that tissue tension would be increased.

During the past several years it has been noted that burned patients rarely die during the so-called period of shock (first 48 hours). It does seem, as has been previously suggested,[2] that an inadequate or delayed therapy will result in a diminished urine flow and that the patients who ultimately die (third to 14th day) excrete small amounts of urine during the first several days. Such patients (A. T. and R. J.) also subsequently show a failure to excrete water and salts in a normal fashion. Thus, if toxic products are formed following a burn, the ill effects produced would be dependent upon the amount of such products present (severity of third-degree burn) and the ability to remove them (kidney and liver function). It would, therefore, seem that if the mortality rate of the severely burned patient (over 30 per cent of the body surface area involved by a third-degree burn) is to be reduced, prompt and adequate therapy should be employed so that an excellent urine volume is assured. In our experience it has been difficult to get a good urine flow subsequently if a good volume is not excreted during the first day. R. J. was given 350 cc. of concentrated plasma (10 Gm./100 cc.) three and one-half days after the burn in an attempt to increase his urinary flow, but the per-minute rate of excretion was not altered. Subsequently distilled water and later whole blood was given for the same reason, but at no time did the rate of urine excretion increase as it would have done in a normal individual.

From the results presented it can be seen that wide differences occurred in the hematocrit during the period of shock which could not be correlated with the extent and depth of the burn. Some patients (Cases 3 and 8) with mild to moderate burns showed higher hematocrits than individuals with severer burns (Cases 9 and 10). On the other hand, some severely burned patients have shown relatively slight changes above the so-called normal value (45). The hematocrit, therefore, is frequently a poor guide to the type and amount of treatment that is necessary, especially since it is influenced by a preëxisting anemia, the rate and number of cells destroyed or trapped from the general circulation at and following the time of injury, and because the normal hematocrit varies quite widely. The first-aid method proposed by Harkins[20] for the control of burn shock (*i.e.*, 50 cc. of plasma for every per cent the body is burned) seems to be more logical, but, again, some patients (Cases 2, 4, 5, 7, 10, 14 and 15) need therapy and yet because of a preëxisting anemia whole blood would appear to be more beneficial than plasma.

In giving treatment for the first 48 hours, therefore, it seems logical to give approximately 50 cc. of blood to adult patients for every per cent the body surface area is burned or to give it in amounts equal to from 1–5 per cent of the patient's body weight in kilograms. In employing the electrolyte

solution it seems desirable to give a slightly hypotonic solution (two-thirds to three-quarters strength in order to increase the rate of urine excretion) in amounts equal to about 10–15 per cent of the patient's body weight during the first two days following the burn. Since little experience has been had with this form of treatment no absolute rules should be employed, but the treatment should be modified depending on the severity and the degree of the burn and the size and response of the patient. When shock is not severe treatment may be given relatively slowly, but if the patient shows evidence of peripheral vascular collapse, therapy should be carried out rapidly until the condition has improved, and urine is being excreted. It should be remembered that the larger quantities advocated above are comparable to giving amounts of fluid which are roughly equal to an individual's total extracellular fluid volume (plasma and interstitial fluid volume), hence, when such amounts are employed, additional liquids and foods should not be given, or should be permitted only in small quantities for the first two days, as this would lead to an excessive fluid intake. If it seems desirable, dextrose can be added to the fluid to provide calories (or some form of protein could be added to the electrolyte solution). In severely burned patients it has seemed that high caloric and protein intakes are better avoided initially.[3] It has been our practice recently to give 800 to 1,000 calories in the form of dextri-maltose daily for the first several days and to supplement vitamins two to five times the normal daily requirement. In mildly burned patients very little blood, plasma or salt solution may be necessary and, thus, a normal diet can often be instituted early.

In most of the patients, excessive hemoconcentration was not encountered when whole blood was given providing an adequate amount of salt solution was employed. In R. J. the hematocrit rose to 76.1, but the patient did not seem to have any ill effects from this rise. This marked increase was apparently due to the giving of blood while the patient was not absorbing adequate amounts of the electrolyte solution due to vomiting.

It has been pointed out[21] that the viscosity of blood is dependent on many factors and that the accumulation of various plasma protein fractions play just as important a rôle as does the addition of red cells. Thus, it seems that if whole blood and an electrolyte solution are simultaneously employed it should not result in a great change in blood viscosity, because as the cell volume increases there is a decrease in the protein fraction. In fact, if large amounts of plasma are given and the albumin fraction is lost, while globulin and fibrinogen are retained (due to the relatively small molecular weight of albumin as compared to globulin and fibrinogen) the blood viscosity might be increased. However, here again, the small increase in the amounts of globulin and fibrinogen would probably not alter the viscosity appreciably because, while they have a much higher viscosity coefficient than does the red cell, the decrease in the cell to plasma ratio produced by the administration of plasma would tend to compensate for the increase in viscosity due to the protein fractions.

When the hematocrit is definitely elevated (55 to 60, or above), the patient's blood pressure is low and little urine is being excreted, we see no reason why plasma should not be given instead of whole blood if it seems to be desirable. In these studies no ill effects have so far been noted if the hematocrit has risen above these levels, but there seems to be no advantage in having an hematocrit of 60, or above, and possibly there is some danger. By the addition of red cells more oxygen might be carried to the tissues and hence anoxia prevented, but it probably is not wise to increase the proportion of cells too much or the beneficial effect will be overcome due to stasis and a slowing of the circulation. Therefore, if hemoconcentration is not excessive it probably would be preferable to give whole blood intravenously and an electrolyte solution orally; but if the hematocrit rises to 60 or above, plasma or a concentrated albumin solution should probably be temporarily substituted for blood. Since several patients have seemingly been taking fluids well by mouth and after two to five hours have vomited, it would seem that where this condition exists the intravenous administration of the electrolyte solution should be temporarily resorted to.

CONCLUSIONS

1. The alterations seen in the hematocrit and in other blood constituents following a burn have been discussed.
2. The effect of various forms of therapy on the blood chemistry has been presented.
3. From this study it would appear that the early administration of whole blood plus an electrolyte solution orally to burned patients is an effective method of combating shock, and that this form of therapy will alleviate the anemia which is usually seen during the period of convalescence.
4. It is felt that the hematocrit cannot be employed as a reliable guide to the amount and type of fluid necessary for treating burned patients.

We are very much indebted to Dr. E. Muntwyler, Professor of Biological Chemistry, Long Island College of Medicine, Dr. F. A. Coller, Department of Surgery, University of Michigan, and Dr. C. A. Moyer, Director of Surgery, Eloise Hospital, for their helpful suggestions and criticisms, and to Mary Allison, Georgiene Eberly, Richard Iwata, Catherine McKay, Jean Reed, Lindley Stout and William Sugiyama for their technical assistance.

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THE RATIONALE OF WHOLE BLOOD THERAPY IN SEVERE BURNS*

A CLINICAL STUDY

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INTRAVENOUS THERAPY is so well-established in the treatment of burns there is no longer reason to question its value, but there remains some question as to the best fluid to use. In 1923, Underhill demonstrated the value of sodium chloride solutions in the treatment of burns and, since 1938, blood plasma has been used on a large scale and has proved of value; recently, the publications of Rosenthal,[1] and Fox,[2] have renewed interest in electrolyte solutions.

The purposes of this paper are (1) to call attention to the red blood cell deficit in the burn patient soon after the burn has been received; and (2) to offer clinical evidence that whole blood may be given advantageously to severely burned patients in the presence of so-called hemoconcentration.

Clinicians have observed that many burn patients given plasma in adequate amounts will show by the fourth or fifth day a moderate to severe anemia. Harkins[3] points out that Schrievers found in experimental studies in burns on rabbits an early reduction in the plasma volume accompanied by a decrease in the total circulating red cell mass. The excellent research of Moyer, Coller, Iob, Vaughan and Marty[4] demonstrated that in the severely burned dog defibrinated whole blood was more effective in controlling burn shock than was plasma, especially if it was given in conjunction with orally-administered sodium chloride-bicarbonate solution.

THE RED BLOOD CELL DEFICIT IN BURNS

Between 1941 and 1944 we had collected a considerable body of blood volume data on severely burned patients. The initial blood volume determinations were made before any fluid had been given intravenously and at 18- to 24-hour intervals for four to five days. Our original interest in blood volume determinations in burn patients was aroused by doubt as to the reliability of estimates of plasma loss when calculated by formulae proposed by Elkinton, Wolff and Lee,[5] and by Harkins[6]; this will be discussed in a later paper. We were further stimulated to reëxamine this blood volume data by the studies of Moyer, *et al.*, on the value of whole blood therapy in experimental burns. When calculations from these blood volume data were made for

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This study was carried out under a contract, recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and the Medical College of Virginia.

total circulating red cell mass in burn patients, it was found that in some of them there existed, soon after the burn had been received, a serious deficit in red cell mass. In nine calculations taken at random from the blood volume data of some of the more seriously burned patients it was found that the red blood cell deficit averaged around 40 per cent of the total volume in deficit. This is shown graphically in Chart 1. It is evident that there may exist in the severely burned patient a serious red blood cell deficit and that this can only be corrected by the administration of whole blood. When only plasma was administered a serious secondary anemia rapidly developed.

We have confidence in the validity of these data on the red blood cell deficit in the severely burned patient for two reasons: (1) When intravenous fluid therapy consisted only of plasma, and redeterminations of the total mass of circulating red blood cells were made at intervals during the first 72 to 96 hours, there seemed to be little change in the size of this mass. If any change occurred, it was in the direction of a further decrease; and (2) if only plasma was given, and the initial data showed a smaller than normal circulating red cell mass, when the blood volume was returning to normal around the 72nd hour there occurred regularly a moderate to severe secondary anemia, which persisted until red blood cells were given.

Other than a deficit in the red cell mass soon after the burn is received the cause of the early anemia in burn patients has not been disclosed by any studies made by us. We do not believe intravascular hemolysis is responsible for the major portion of this red cell deficit because in many patients there was little or no staining of the plasma with free hemoglobin; we are of the opinion that the red cell deficit is due in the main to sludging or trapping of large masses of red blood cells in the capillaries in and adjoining the burned area. This view is supported by the work of Moritz[7] who found quantities of iron in the skin of experimentally burned animals, amounting in some cases to around 30 per cent of the total circulating hemoglobin.

It might be stressed that hematocrit data do not necessarily indicate the extent of the anemia in burns, especially when plasma and red cells are lost in disproportionate amounts into the burned area. Further, some of the deficit in red cell mass, especially in children and women in our series, may have been the result of a preëxisting anemia. This view is supported by the findings of Dr. T. Stanley Meade,[8] who has found a moderate to severe secondary anemia in approximately 50 per cent of the children examined by him in our local health clinics.

CLINICAL STUDIES OF WHOLE BLOOD THERAPY IN SEVERELY BURNED PATIENTS

The main purpose of this study was to evaluate whole blood transfusion* in the management of burn shock in the severely burned patient. Since our studies on red cell deficit in the burn patient (mentioned above) indicated that without blood volume data the hematocrit reading might give no indication of

* By whole blood transfusions we mean "citrated blood"; 50 cc. 5 per cent sodium citrate is used as anticoagulant in each 500 cc. whole blood.

red cell loss, it was decided to give whole blood transfusions to this series of patients regardless of hematocrit readings. We were encouraged to do this, despite the height of some hematocrit readings, by the report of Moyer, *et al.*, that scalded animals with hematocrit readings of 75 to 80 per cent could be given massive transfusions of defibrinated blood, and that these were compatible with life. Accordingly, whole blood transfusions were given in some cases when the hematocrit readings were as high as 65 to 67 per cent.

The general plan of a clinical experiment was to treat the patient's burn by pressure dressings and to give intravenous whole blood infusions of 500 to 1,000 cc. every six hours for the first 48 hours, along with enough saline and other fluids to keep up a urinary output of 50 to 100 cc. per hour. Every effort was made to have the patient take fluids by mouth rather than by the intravenous route. Fluids were given in the form of water, soft drinks, milk, or fruit juices. This required special nursing care, which was provided in most instances. No attempt was made to give large amounts of sodium salts, orally or intravenously, but with each whole blood transfusion 8 Gm. of sodium bicarbonate was given. Some patients who took fluids best in the form of soft fountain drinks, such as coca-cola, were allowed to have them. All patients were typed and given only type specific blood. At the suggestion of Dr. Philip Levine,[9] all female patients were given *Rh*-negative blood until it could be demonstrated that this was unnecessary. For children one to five years of age the six-hourly infusions of whole blood were usually limited to 150 to 200 cc.; in all burned patients every effort was made to give whole blood to the amount necessary to keep the hemoglobin level above 100 per cent during the first four days of therapy. Penicillin, usually 100,000 units daily, was given to all patients.

In this series of 32 burn patients there have been three deaths: The first, an eight-year-old Negro child, was found in a burning house in which her mother and sister perished. The child was brought to the hospital about four hours after having been burned and was in a state of severe shock; the blood pressure being unobtainable. There were third-degree burns of approximately 60 per cent of the body surface, with severe burns of the face. When the bladder was catheterized no urine was obtained. The initial hematocrit was 67 per cent. She was given during the next eight hours approximately 1,000 cc. of whole blood and 1,000 cc. of saline. In four hours 470 cc. of urine was obtained. The child died following generalized convulsive seizures about eight hours after admission. At catheterization of the bladder after death 210 cc. of urine was obtained. The nonprotein nitrogen was 26 on admission and 40 shortly before death. It would appear that in this apparently hopelessly burned child, admitted in a state of severe shock, whole blood therapy at least resulted in a return of renal blood flow to well above that usually found at the shock level. The second death is that of a 57-year-old Negro male, with approximately 80 per cent body surface involved by third-degree burns. He had been found lying in a bed that had caught fire, and had been apparently stuporously drunk at the time. On admission, he was

unconscious, with a blood pressure of 70/50. He was given during the next four hours approximately 2,000 cc. of whole blood and equal quantities of normal saline, but died four hours after admission. The third death occurred in an 11-year-old burned child, 26 days after admission. This patient's treatment and course will be commented on in detail (Case 1).

CASE REPORTS

Case 1.—(Fig. 1, Chart 2): C. P., age 11, was admitted to the Medical College Hospital, January 5, 1945, after having suffered a severe burn. While standing in front of a bonfire his clothing became ignited. The flames were finally smothered by blankets. On admission to the hospital, there was a deep burn extending from the nipple line over the entire body down to the ankles. Sedation was secured with intravenous morphine gr. 1/6. The burned areas were cleaned with soap and water and dressed according to the pressure dressing technic. Fluid therapy was given as shown in Table I. During the first 24 hours the patient received 2,500 cc. of blood and adequate fluids by mouth so that a urine output of approximately 2,000 cc. was secured for that period. Reference to Chart 2 illustrates that this patient received whole blood transfusions despite the presence of what is considered severe hemoconcentration. During the second 48 hours he received an additional 1,000 cc. of whole blood and adequate fluids by mouth to maintain a good urinary output. The child was quite ill during the first four days of hospitalization, but by the 96th hour showed marked improvement. He was then able to take all necessary fluid by mouth and maintained daily a good urinary output. Penicillin was given every fourth hour, 10,000 to 15,000 units. The first dressing was done on the 12th hospital day, at which time the photograph shown in Figure 1 was made. At this dressing it was observed that sloughing would take place of the full-thickness of the skin from the ankles to the axillae. At this time, however, the child was in remarkably good condition. He was given whole blood transfusions to the amount of 500 cc. about every third day after the first four days. The hemoglobin levels, which were taken daily, never fell below 82 per cent, and generally were above 90 per cent. The plasma protein, while it was 5.4 per cent on the fourth day, steadily rose, so that by the tenth hospital day it was 6.2 per cent, at about which level it remained. In addition to the blood transfusions a daily intake of approximately 100–120 Gm. of protein was maintained. Although we despaired of ever securing enough skin for grafting, the child's condition appeared hopeful until January 29, the 24th hospital day, at which time he became irrational. Blood chemical studies were all within the normal range, the nonprotein nitrogen being at no time above 46 mg. per cent. It was noted on January 29 that the blood pressure was rising, and late on that day the readings were regularly 160/110, whereas previously they had been in the range of 110/70. Gradually a comatose state set in, and by January 30 the child could not be aroused. All attempts at plasma, whole blood, sodium chloride or sodium lactate therapy failed to change the condition. Despite every attempt at therapy the child died on January 31, 1945. A postmortem examination was secured. This examination showed the burned skin to involve approximately 60 per cent of the body surface. The autopsy revealed no adequate explanation for the patient's death; since permission for examination of the brain could not be obtained, it is impossible to eliminate an encephalitis or degenerative cerebral lesion as a primary cause of death (see Walker and Shenkin[10]). Sections of the liver and kidneys showed no changes from the normal.

COMMENT: This burn patient represents one of the most serious we have treated, and was, indeed, a real test for the efficacy of whole blood transfusions for the management of burn shock. The fact that the child was kept alive for 26 days, with fairly normal levels of hemoglobin, plasma protein and

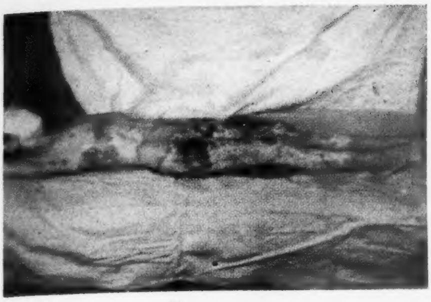

FIG. 1

FIG. 2

FIG. 3

FIG. 4

FIG. 5

FIG. 6

FIG. 1.—Case 1: C. P., taken on first redressing on the 12th hospital day. Full-thickness burns from ankles to nipple line.

FIG. 2.—Case 2: D. T., illustrating extent and depth of the burns of the legs. Figures 7, 8 and 9 show the extent of the burns in this patient of the left arm, thorax, abdomen and buttocks.

FIGS. 3 and 4.—Case 3: H. R., illustrating extent of the burns. Photograph taken at first redressing.

FIG. 5.—Case 4: W. H., taken at first redressing

FIG. 6.—Anterolateral portion of the thorax of badly burned child taken at the time of first grafting. The good state of nutrition is to be noted. This child received large and frequent whole blood transfusion at the time of entry.

TABLE I

FLUID BALANCE SHEET FOR PATIENTS DISCUSSED IN DETAIL IN TEXT

	Oral				
Day	Water	Milk	Fruit Juice	Intravenous	Urine
Case 1. C. P.					
1	1600	550	510	2500 blood 2000 saline 1400 water 3 amp. sod. lact. 32 Gm. $NaHCO_3$	1735
2	2445	710	215	500 blood 500 saline 2 amp. sod. lact. 8 Gm. $NaHCO_3$	1975
3	1730	1040	90	500 blood 1500 saline 1 amp. sod. lact. 8 Gm. $NaHCO_3$	1985
4	1420	1640	680		2745
5	1515	2235	680		1625
Case 2. D. T.					
1	1560	....	500	3300 blood 3000 saline 2500 water 2 amp. sod. lact. 32 Gm. $NaHCO_3$	1525
2	2370	720	790	2000 blood 2500 water 1000 saline 1 amp. sod. lact. 32 Gm. $NaHCO_3$	2010
3	3240	1050	200	500 blood 8 Gm. $NaHCO_3$	1795
Case 3. H. R.					
1	1470	....	400	2500 blood 1000 water 1000 saline 2 amp. sod. lact. 24 Gm. $NaHCO_3$	1900
2	360	....	250	1500 blood 3000 water 24 Gm. $NaHCO_3$	2100
3	1210	200	650	500 blood 8 Gm. $NaHCO_3$	1000
4	2460	360	380	3000 saline	1350
Case 4. W. H.					
1	1860	300	660	2500 blood 500 saline 1 amp. sod. lact. 32 Gm. $NaHCO_3$	2762
2	1860	360	500	1000 blood 16 Gm. $NaHCO_3$	2400
3	1540	450	640	500 blood 8 Gm. $NaHCO_3$	1900
4	1280	480	440		1200
5	1430	720	560	500 blood 8 Gm. $NaHCO_3$	1300

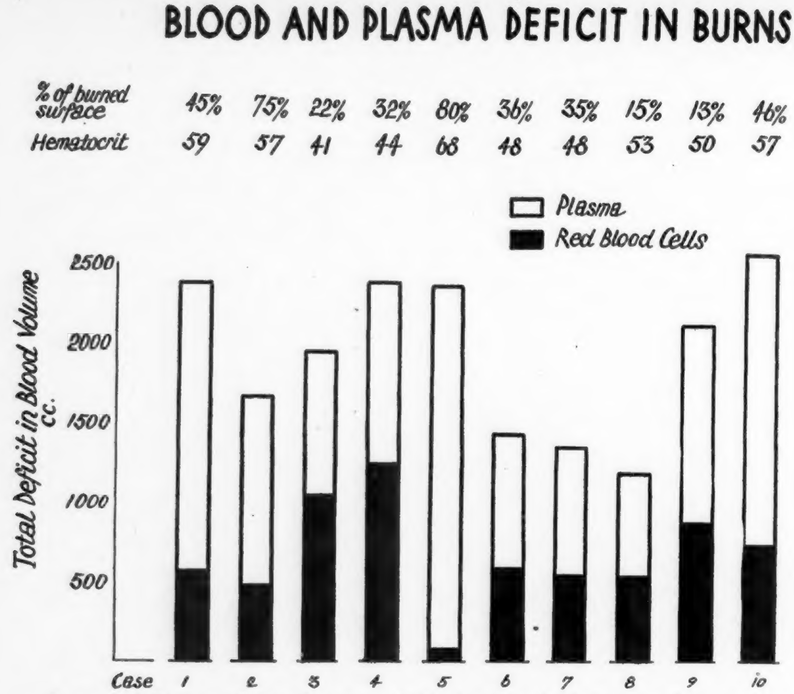

CHART 1.—Diagram illustrating the red blo d cell and plasma loss in a series of severely burned patients. Calculations were made on the basis of a normal person having a total blood volume of 80 cc. per Kg., and an hematocrit of 45%. Cases 2 and 5 died on the fourth and fifth days, respectively; the other patients survived. Case 5 was largely a second-degree burn.

Fluid intake	8460	4270	5160	
Urine	1735	1975	1985	
Total protein	5.2%	5.2%	5.4%	5.4%
NPN	38	32	46	43

CHART 2.—The treatment and course of Case 1, C.P.

urinary output attest, we believe, to the value of whole blood transfusions in the badly burned patient.

Case 2.—D. T., white, male, age 27, was admitted November 18, 1944. In a fire which totally destroyed an automobile trailer (in which his wife and child perished), the patient suffered severe deep burns of left arm, thorax, trunk, buttocks and legs, as shown in Figures 2, 7, 8 and 9. On admission, the blood pressure was 115/70. Pressure dressings were applied rapidly, and intravenous blood and saline therapy begun. Thirty-three hundred cubic centimeters of whole blood was given during the first 24 hours, and 2,400 cc. during the second day. Whole blood was given in the presence of marked

Fluid intake		10,860	9380		4490
Urine		1525	2010		1795
Total protein		5.0%	5.2%		5.4%
NPN	50	90	40	58	44

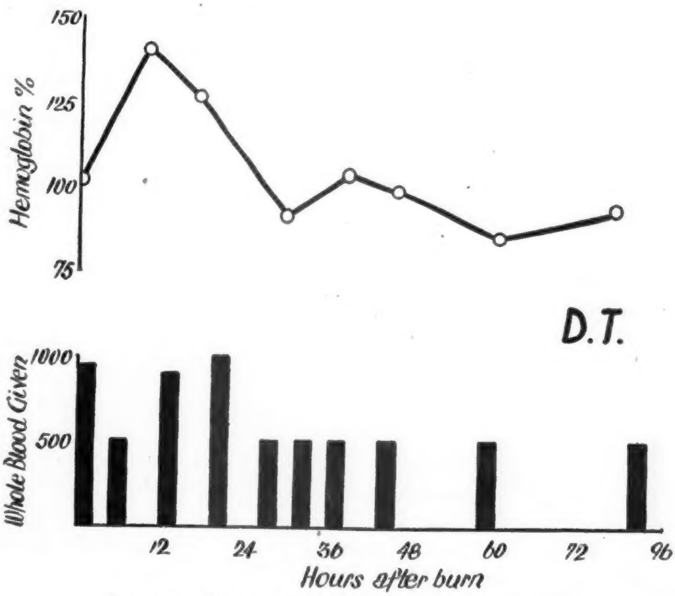

CHART 3.—The treatment and course of Case 2, D. T.

hemoconcentration in this patient (Chart 3). Urine output was good, although the nonprotein nitrogen rose to 90 at the 12th hour, returning to 40 the next morning. Fluids were taken well by mouth, and on the third day a special high protein diet was started which the patient took well. The first redressings were made of the arms and chest on the 14th day, the buttocks and legs five days later. Grafting of the legs was done first on January 2, 1945. It is interesting to note that this extensively burned patient maintained a good hemoglobin level (never below 79 per cent during his six-month hospital stay); this we attribute to his excellent coöperation in taking a special high protein diet daily during his first five months of hospitalization and which will be discussed in a later paper. He was discharged June 3, 1945, after grafting and intensive physiotherapy, able to walk. He will return later for revision of a graft of the left popliteal space.

Fluid intake	6370	5110	2560	6200
Urine	1900	2100	1000	1350
Total protein		6.2%	6.4%	
NPN		80	42	40

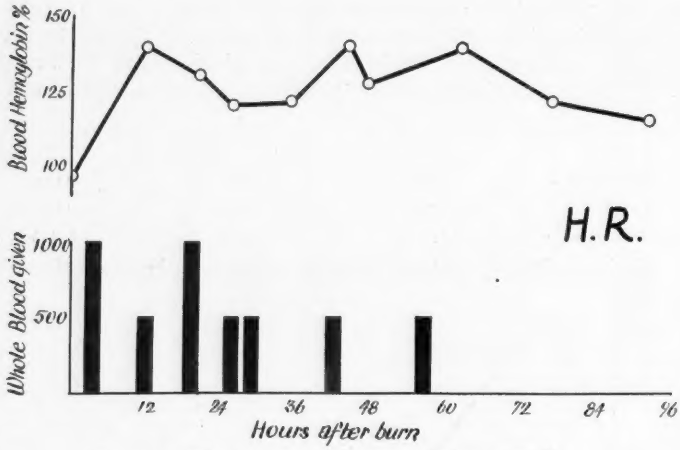

CHART 4.—The treatment and course of Case 3, H. R.

Fluid intake	5820	3700	3130	2200	3210
Urine	2762	2400	1900	1200	1300
Total protein	6:2%		5.8%		6.0%

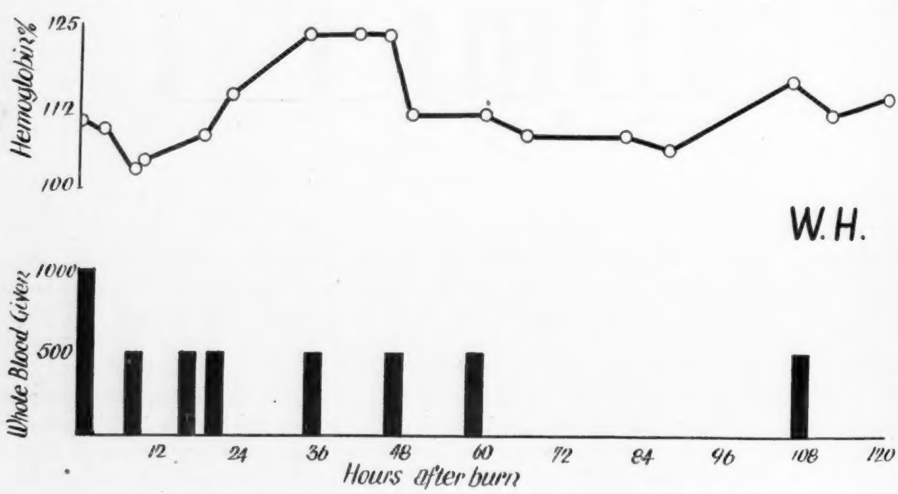

CHART 5.—The treatment and course of Case 4, W. H.

FIG. 7

FIG. 8

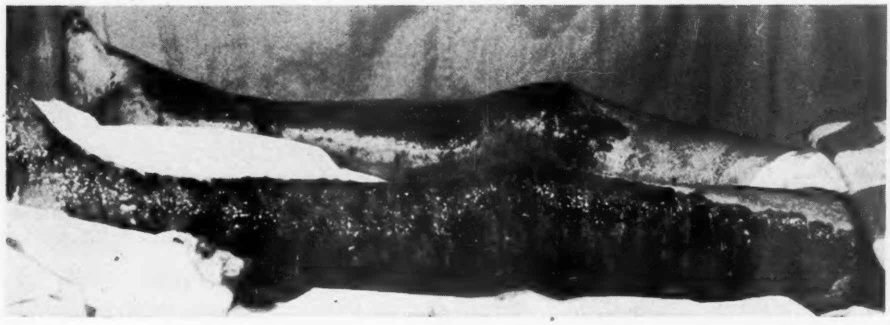

FIG. 9

FIGS. 7, 8 and 9.—Case 2: D. T., showing extent of burns. Figures 7 and 8 taken at first redressing on the 14th day. Figure 9 taken four weeks after the burn had been received. In extensive burns of this nature, it is our practice to redress only small portions of the burned surface at one session.

Case 3.—H. R., a 34-year-old Negress, was admitted to St. Philip Hospital January 17, 1945. Her clothes caught fire while she was "throwing kerosene into the stove." Despite extensive burns as shown in Figures 3 and 4, the patient was in good condition on admission. Pressure dressings were applied, and the patient given whole blood transfusions as shown in Chart 4. She took fluids well, and maintained a satisfactory urinary output. Her clinical course was excellent; the first redressings were made on the 14th day, at which time photographs shown in Figures 3 and 4 were made. Although her hemoglobin levels were kept above 85 per cent during the first 29 days of hospital stay, and while she was taking well a special high protein diet, her clinical course was marred by the fact that vigilance relative to diet was relaxed, so that during the second month of hospital stay the hemoglobin dropped to 70 per cent. This was corrected by 1,500 cc. whole blood infusions and resumption of the special diet. Excellent healing occurred of all badly burned parts except the under portions of forearms; these were covered with split-thickness grafts.

Case 4.—W. H., an 18-year-old male Negro, who suffered burns of thorax and abdomen, hands and forearms, thighs, scrotum, and penis, was admitted to St. Philip Hospital January 6, 1945. He received his burns as a result of throwing motor oil on an open fire. On admission, the patient was in good condition, and pressure dressings were applied in the ordinary manner soon after entry. He was given 2,500 cc. whole blood during first 24 hours, and 1,000 cc. whole blood the second day (Chart 5). His hemoglobin remained in the neighborhood of 110 per cent after the first 48 hours. Healing of the burned areas was extraordinarily rapid, the photograph shown in Figure 5 being taken on first redressing on the 14th hospital day. Plasma protein levels never fell below 5.8 per cent, and after the 14th hospital day remained above 6.8 per cent. The special high protein diet was given starting on the fourth hospital day. During the first month of treatment the hemoglobin was consistently 100 per cent or above, although no more blood transfusions were given after the fifth hospital day. On February 2, 1945, the penis was covered with a dermatome split-thickness graft and "postage stamp" grafts, cut with the dermatome, were applied to the left thigh. The "take" was 100 per cent. The patient was discharged March 1, 1945, with complete recovery.

THE CLINICAL EVALUATION OF WHOLE BLOOD THERAPY FOR BURN SHOCK

The besetting sin of clinical investigation can be preference for argument over observation

After treating several hundred burn patients, we find it difficult to compare the effectiveness of blood and so-called blood substitutes, one with another. It is, therefore, difficult for us to compare whole blood with plasma in the management of burn shock. For this reason some of the observations cited below concerning the value of whole blood in the initial treatment of the severely burned patient might best be termed "clinical impressions." They should not be construed as detracting from the value of plasma in burn therapy.

(1) *Blood Hemoglobin Levels:* In our experience, if fairly large amounts of whole blood are given to the burn patient during the first 48 hours "masked anemia" is not encountered, whereas in burn patients treated only with plasma this condition occurs frequently. It appears, therefore, that if whole blood is given in adequate amounts during the burn shock period secondary anemia will be prevented. This leads us to believe that secondary anemia in burn patients may be more easily prevented than treated.

(2) *Plasma Protein Levels:* When burn patients are given plasma alone, even in large amounts, it is not unusual to find low plasma protein levels on the fourth or fifth day. This is especially true if the burn is deep and extensive (above 40 per cent). On the other hand, in this series of burns treated with large amounts of whole blood the plasma protein levels were maintained at more nearly the optimum level (Cases 1 to 4). This may be due to improved blood flow through the liver during the burn shock period and, therefore, less liver anoxia in the whole blood treated patients. Stated differently, one of the advantages of whole blood therapy may be the maintenance of the liver in such a state that plasma protein production is carried on in a more nearly normal manner during the initial burn period. It should be remembered, however, that this group of patients received large and frequent transfusions.

(3) *Urinary Output:* As was noted in the detailed case reports, we have found it possible to maintain a good urinary output even when hemoconcentration appeared to be quite marked. It is evident from the nonprotein nitrogen figures that renal blood flow must have been maintained at a level adequate for good renal clearance. Difficulty on this score was encountered with only one patient (Case 5).

Case 5.—A 57-year-old colored female, M. T. [on whom we have previous clinical records indicating that she was suffering from hypertensive cardiovascular disease (B. P. 230/140, retinal changes Grade 3)], when she received a deep burn of approximately 25 per cent of the body surface on March 1, 1945. She was treated with whole blood infusions totaling 2,500 cc. during the first 48 hours, and maintained a good urinary output for the first three days (1,900, 1,725 and 1,285 cc., respectively). Her hemoglobin levels ranged from 100 to 120 per cent during this time. On the fourth day, when her hemoglobin level was 106 per cent, the blood nonprotein nitrogen rose to 106, and her urinary excretion diminished to less than 600 cc. From then on she was given only approximately 2,000 cc. of fluid by mouth each day. By the seventh day the nonprotein nitrogen had come down to 46, and urinary excretion was good, and remained so.

An attempt should be made to induce the burn patient to take fluids and food (especially protein) *by mouth,* so that it is not necessary to give large amounts of fluid intravenously. The observations of Moyer, *et al.,* emphasize this point forcibly. We have hesitated throughout our studies on burns to employ excessively large amounts of fluid by vein precisely for the reasons pointed out by Moyer. Conscious patients, given expert nursing care, will take adequate amounts of fluid by mouth, especially if given the type of fluid they desire. The first burn patient we treated with plasma in 1939, would take only one particular soft drink but drank six to eight bottles of it each 24 hours for the first two or three days. By letting patients have fluids of their own preference and at intervals according to their wishes, or after quiet but firm insistence by the nurse, it is usually possible to maintain a good urinary output throughout the 24-hour period, but when intravenous fluids are resorted to, one usually finds that good urinary output occurs only during and shortly after the fluid administration.

(4) *Toxemia:* Our clinical impression is that these burn patients treated

with whole blood have shown less "toxemia" than did other patients treated with plasma or gelatine. This can only be an impression. The temperature and pulse curves of the four patients whose records were presented above are: Case 1, C. P., for the first four days rectal temperature ranged between 100° and 102° F., pulse rate between 90 and 120; Case 2, D. T., for the first four days rectal temperature ranged 102° to 104° F., pulse rate consistently around 120; Case 3, H. R., for the first four days temperature ranged from 100° to 102° F., pulse rate 90 to 120; Case 4, W. H., rectal temperature first four days 100° to 103° F., pulse rate 100 to 110. The respiratory rates of all four patients averaged around 30 for the first four days. It should be recalled, however, that Case 1, C. P., was at times irrational during the first two days and vomited small amounts at intervals during this time. One other patient, a badly burned four-year-old colored child, appeared to be quite toxic during the first four days of therapy.

(5) *Healing of Burns:* This is another problem that defies comparison of one series of burns with another because it is difficult to judge the exact depth of the burn in an individual patient, but, in general, burns caused by actual fire are deeper than those caused by hot water or steam. It may be significant, therefore, that in this series all patients were burned by fire. On the basis of our former experience one would have expected that more or less extensive grafting would be required in many of the patients in the present series; such was not the case.

We realize that no known therapy will convert a full-thickness burn into one that heals satisfactorily without grafting. Nevertheless, in burns with severe destruction of the tissues, which heal without grafting, there are several factors that might act to promote or retard epithelial growth. For example, if in a burned area viable epithelial cells are left at the base of hair follicles or sweat glands, healing may take place by outgrowth from these "hidden islands" if (1) the dressing is left undisturbed for long periods of time; (2) infection does not supervene; and if (3) proper nourishment is available to promote rapid growth in the residual epithelial cells.

We have been pleased with the rapidity with which growth of epithelium has taken place in burned areas that at first appeared of a depth and extent to require extensive skin grafting. We do not wish to overemphasize the importance of adequate amounts of whole circulating hemoglobin in the healing process but only with an adequate circulating red cell mass can oxygen and food, such as amino-acids, be carried to the zone of injury in sufficient quantities. With an inadequate number of red cells, healing cannot be rapid.

(6) *Intravascular Clotting:* An increased incidence of thrombosis in burn patients with hemoconcentration has been feared, and one might hesitate to give whole blood for this reason, but it is interesting to note that in this series of 32 patients, 29 of whom survived, there was no instance of thrombophlebitis or pulmonary embolus clinically recognizable.

Finally, it should be emphasized that whole blood has been used in this

series of patients to determine whether it could be given safely to the burn patient in the presence of moderate to severe hemoconcentration. Our experience indicates that whole blood can be given safely under these conditions.

CONCLUSIONS

Blood volume determinations of severely burned patients made soon after the burn had been received indicate a decrease in total circulating red cell mass. It is believed that this initial loss of red blood cells may account for a considerable portion of the "masked anemia" that appears in the post-shock period in many burn patients.

Whole blood infusions have been employed for the management of burn shock in a series of 32 severely burned patients. Whole blood has been given in the presence of marked hemoconcentration. Apparently, whole blood can be given safely to burn patients with hemoconcentration. If adequate amounts of whole blood are given initially in severely burned patients, secondary anemia is regularly avoided.

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DIRECT FLAP REPAIR OF DEFECTS OF THE ARM AND HAND*

PREPARATION OF GUNSHOT WOUNDS FOR REPAIR OF NERVES, BONES AND TENDONS

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GUNSHOT, shell fragment and other types of wounds may leave large surface defects and extensive, crippling scars of the arm and hand. These can be repaired with direct abdominal and chest flaps, by using the principle of a short, broad pedicle, which will allow complete mobilization and immediate use of the flap. There is rarely any need for delaying or tubing these flaps.

This procedure has been carried out in a large number of patients, and a valuable saving of patient-hospital-weeks has been possible. Whereas, long tubed flaps or delayed flat flaps have been known to require months of preparation, this direct type of flap is prepared in 10–30 minutes, and usually can be detached in 14–20 days. So that, the crippled extremity is freed of its scar by thorough dissection, the flap is prepared accurately, at the same time, the arm is "planted" under the flap, and in 2–3 weeks the arm or hand can be detached from the abdomen and the wound closed. It is soon ready for use, or for any necessary deep work on bone, nerve or tendon.

Bone, nerve and tendon repairs cannot be accomplished successfully through dense scar, because the results of these operations can only be as good as the surface healing. When deep repairs are attempted through excessive scarring, the wounds may break down, and wire, foil, screws, plates and bone grafts may be lost. The procedure outlined here is of marked importance in the preparation of many areas for necessary orthopedic or neurosurgical repairs.

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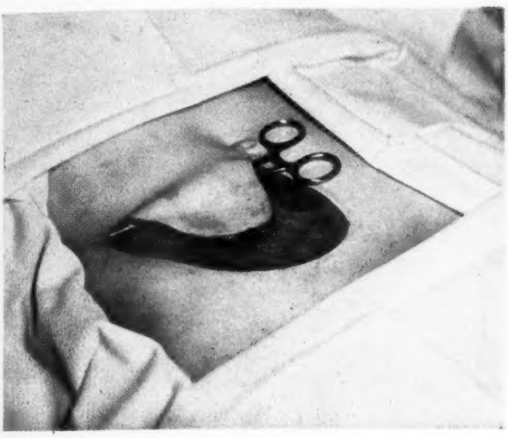
* This article was to have been presented before the Annual Meeting of the American Surgical Association, May, 1945.

A

B



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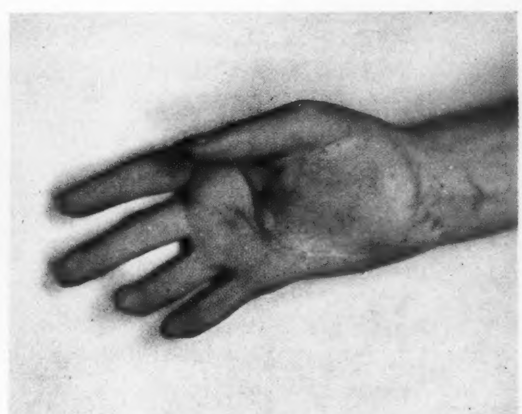


F

FIG. 1.—A to F.—Multiple shell fragment wounds of hand with resultant fibrosis. Widespread dissection with removal of shell fragments encountered. Repair with direct flap detached in 18 days.

"Surface healing can be only as good as the deep blood and nerve supply. Wounds that are excessively fibrotic, and have a diminished blood supply, often cannot maintain the nutrition of their own skin covering, and a newly transplanted surface over such a wound may not survive. This is seen in radiation burns and in chronic leg ulcers. The same conditions are also seen in gunshot and shell fragment wounds where extensively torn tissues have healed (or attempted to heal) in contracted, dense scar masses, the surfaces of which repeatedly ulcerate. Such wounds may have rough, keratosic surfaces. This may be especially true in 'through-and-through'

FIG. 1G.—Roentgenogram of hand shown in Figure 1, Color Illustration.

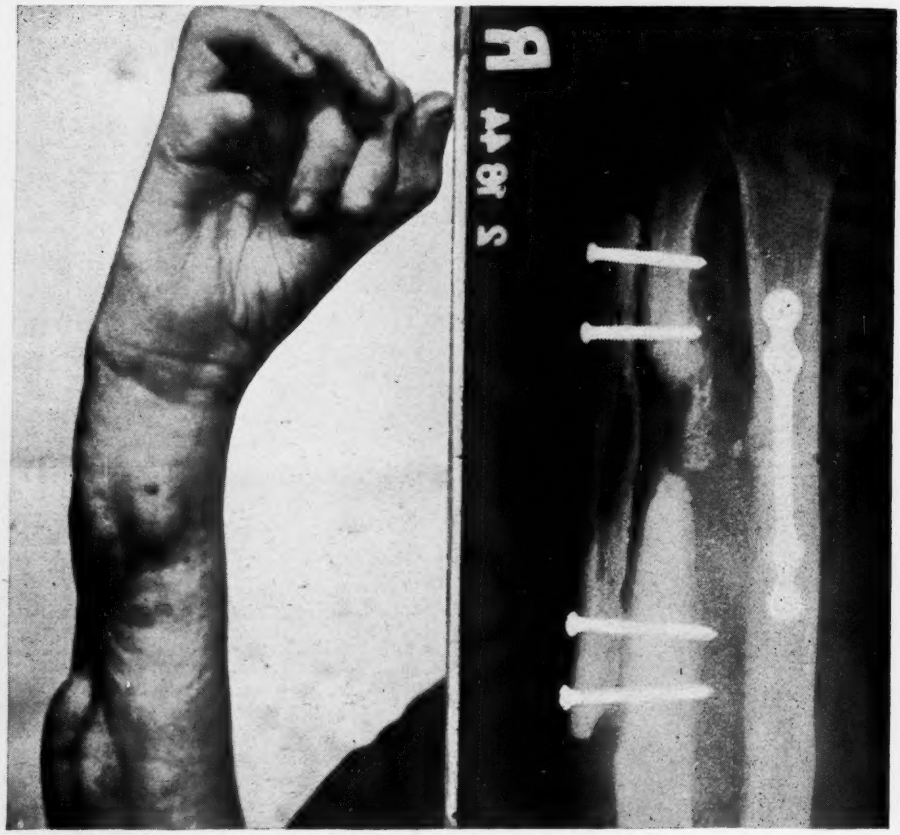
injuries, because the track of scar tissue completely penetrates the area, so that, in a dissection, the surgeon does not encounter a soft, normal bed of tissue.

"The factors to be balanced in planning satisfactory wound closures in preparation for some subsequent bone, nerve, or tendon repair are, briefly: (1) the preparation of deep tissues to carry adequate minute blood supply to maintain the surface repairs, by the resection of surface and deep scar continued into an area that will furnish satisfactory circulation; and (2) the designing of a surface closure through the use of local flaps, skin grafts, or direct or delayed pedicle flaps."[1]

These direct flaps also may be used within the first few days of the original injury, and tendon and bone fragments can be saved and bone union advanced. This is a marked advantage over the plan of letting wounds collapse and heal, with distortion, so that scar has to be resected and tissues

replaced in position when the repair is undertaken weeks later. This has been recorded,[2] and Colonel E. M. Bricker has reported, by personal communication, that many early flaps have been used successfully in the Plastic Surgery Centers in the European Theater.

Diagnosis and *recording* of arm and hand injuries is extremely important, and a separate note for each finger is required. Sensation in fingers is



A B

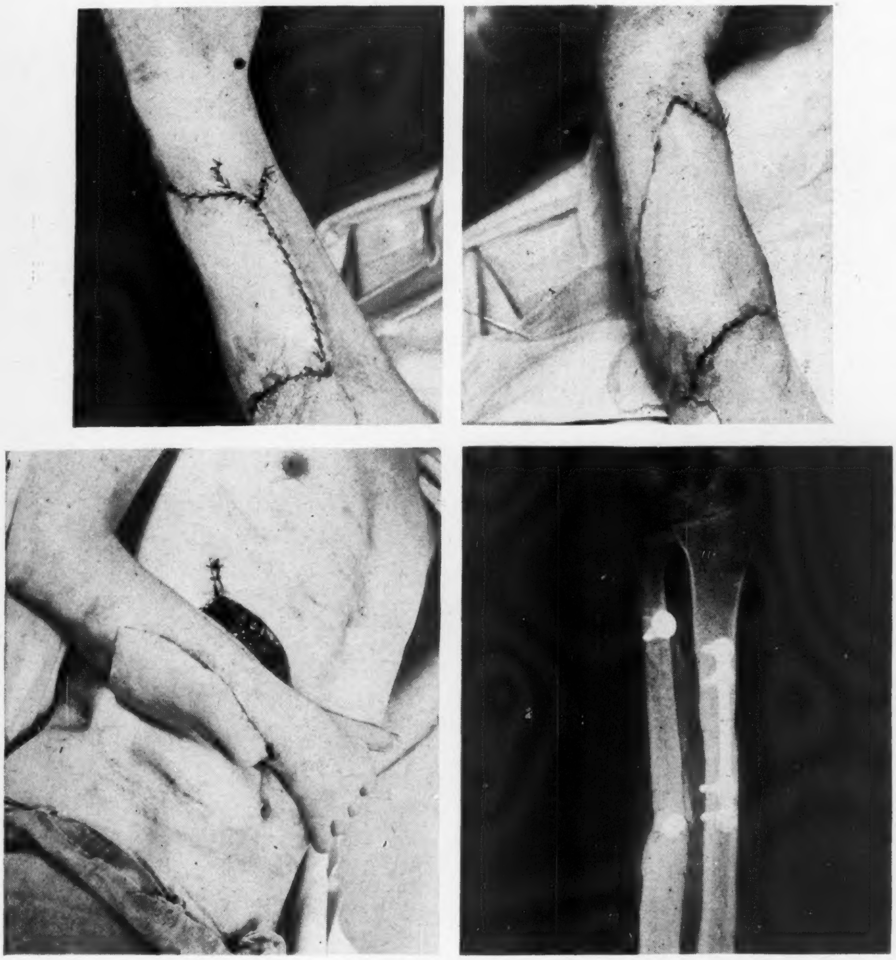
FIG. 2

FIG. 2.—A to F—Shell fragment wound of arm with loss of bone graft from breaking down of the scar. Extensive replacement of soft tissues with direct flap and successful secondary bone graft. A soft-tissue shadow of the flap may be seen also on the roentgenogram.

of paramount importance, and the response to pin-prick is always obtained and recorded. On this point of sensation may depend the decision of trying to save or to remove fingers. This is an evident finding, but one that apparently should have attention called to it frequently. This is often tedious in a flood of patients, many with both hands damaged, but it is *always* done and recorded and the notes referred to in the operating room, because the response cannot be obtained with the patient under an anesthetic.

Simplified Designation of Fingers and Joints.—To avoid repeated use of cumbersome terms such as "the metacarpophalangeal joint of the middle finger" or the "distal interphalangeal joint of the index finger," a simple (if drab) method of recording can be used, with relief and speed, namely, the

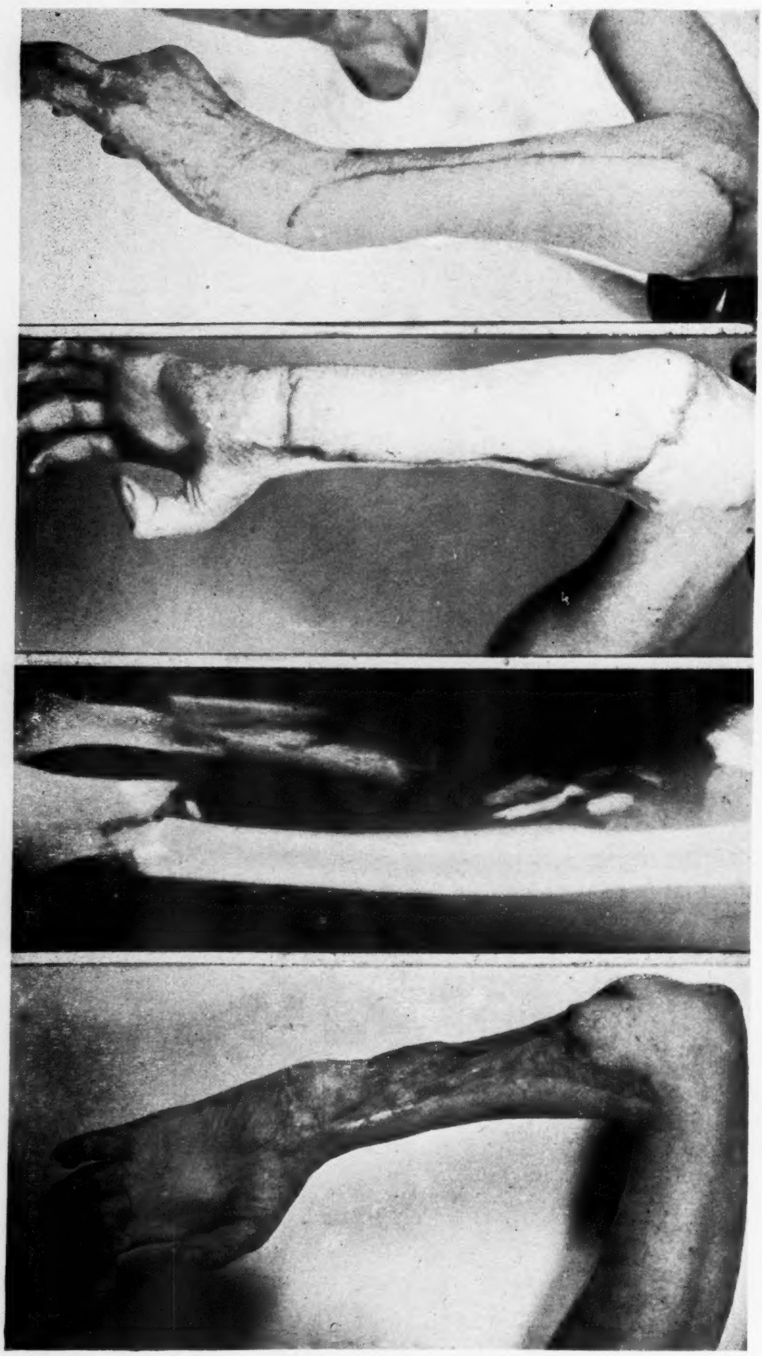
C D



E F

Fig. 2 (Continued)

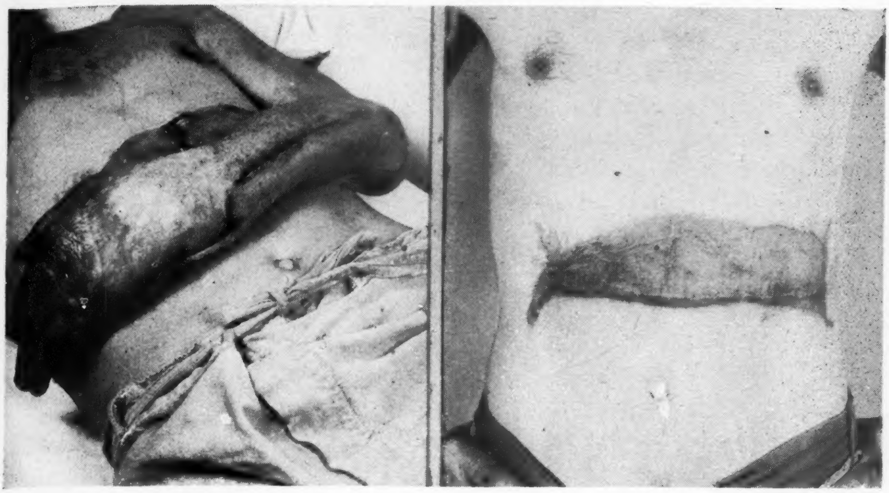
fingers are simply called 1, 2, 3, 4, and 5 and the joints A, B, and C. Thus, the above long designations become simply "3A" and "2C." If desirable the metacarpals and phalanges are called W, X, Y, Z and "the metacarpal of the ring finger" becomes "4W" and "the middle phalanx of the little finger" becomes 5Y. A simple chart can be used, but seldom has to be



A B C D

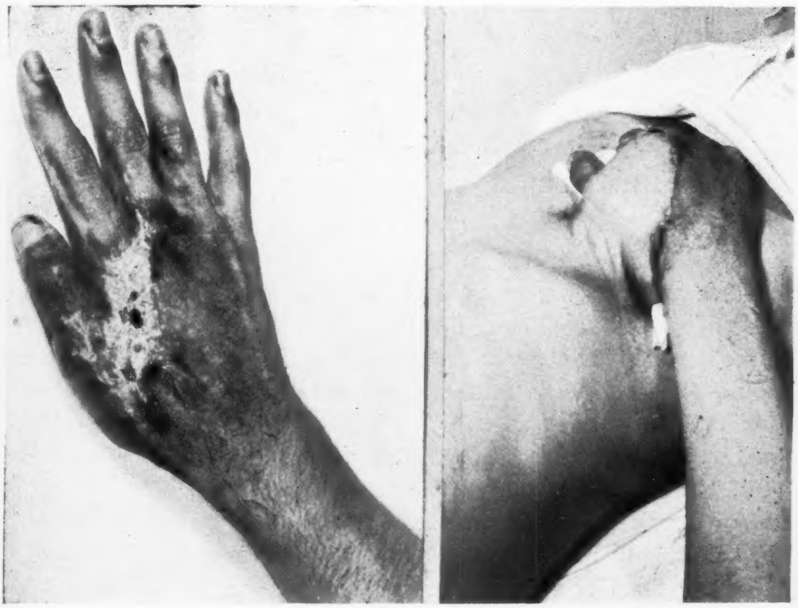
FIG. 3

FIG. 3.—A to F—Extensive loss of soft tissue and destruction of bone showing widespread replacement of soft tissue in 18 days with a direct flap from the chest and abdomen, preparatory to deep bone and tendon work.



E F

FIG. 3 (Continued)



A B

FIG. 4

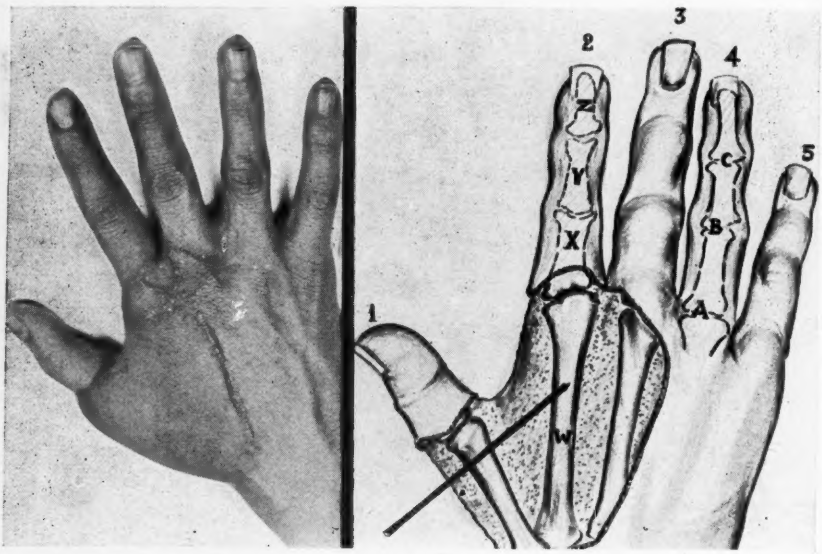
FIG. 4.—A to D—Gunshot injury of hand with a direct dorsal flap after separation of thumb from the fingers. No deep work required.

D shows method of internal pin fixation to fix the thumb into position.

Nomenclature is indicated using 1 2 3 4 and 5 for the fingers, A B C for the joints and W X Y Z for the bones.

referred to. The thumb, of course, has no C-joint or Z-phalanx. This is illustrated in Figure 4.

Technic: The open wound is prepared or the scarred area is resected so that adequate minute blood supply is present all around. (For a defect extending around the arm, there is no use doing the entire dissection if the flap cannot reach until it is taken off later.) If the dissection is done under a tourniquet, it is released and hemorrhage is controlled, before the flap is put on (Fig. 1).



C D

FIG. 4 (Continued)

The flap is located in a suitable, comfortable place on the abdomen or chest. For a hand, it is most often in the lower quadrant on the same side, avoiding pubic hair if possible. For the volar surface, the pedicle is usually up towards the chest, and for the dorsal surface, it is usually down, toward the inguinal region. This is, of course, determined by the freedom of movement of the joints, but it is not necessary to put the hand clear across the abdomen. The position of the upper arm and elbow alongside the body and on level with the bed is the most comfortable for hand flaps.

The flap is raised on the principle of a broad, short base, and is designed to fit the defect, but with an over-all additional allowance for shrinkage, which is roughly one-third (Fig. 1). The pattern material is not important, but pliofilm or celluloid are usually available.

The bed of the flap is usually reduced in size with sutures along the edge, and often the remaining defect is grafted with a split-graft. The closed wound makes for easier convalescence and easier care during it—but it is not an essential.

The flap is sewed in loosely along its base with a few interrupted fine

sutures and then is closed around its two or three sides with deep fine sutures and a few skin sutures.

Firm fixation is obtained with a few large strips of adhesive—not plaster of paris—and a cotton waste pressure dressing is put on that can be turned back for easy inspection of the circulation of the flap.

Pressure dressings on these flaps are very important, to prevent venous stagnation and the inspection should be done an hour after operation and in the evening, and as often as necessary. Gentle pressure will save more flaps than any other procedure providing, of course, that the position is correct and that there are no kinks (Fig. 1).

Throughout the period of fixation the dressings are kept fresh and clean and adequate dressing service is essential; the success of many flaps has been dependent on nurses in the dressing room, and certainly the comfort of the patient has been possible only through their work.

Splints may be necessary to prevent retraction and collapse of hands and they easily can be used while the arm is in place by having neatly cut aluminum splints with or without extensions for elastic traction on the fingers. Traction may be from the fingernails or from skeletal wires. *Internal wires* are often used to fix fingers or metacarpals in position, during the period of attachment if external splinting or fixation will not suffice (Fig. 4).

Thumb rotation is always important and is maintained whenever possible. If rotation is not possible, abduction and extension can be relied on.

Donor areas are selected, as mentioned, for comfort, to avoid pubic hair, to give the best type of skin. On the hand thin skin of the inguinal region or even of the thigh is preferable for fingers and the palm.

Free Skin Grafts versus Flaps.—It is worth noting that on the fingers and on the palm, free skin grafts are preferable to flaps if they can be used; and in *some* patients, even if there is some question, free grafts will be used rather than bulky flaps. Free full-thickness grafts from the neck are valuable on the dorsum of fingers, to obtain the best pad.[4]

Cross arm flaps are used when the good thin skin of the arm is thought to be necessary to secure function in a finger or the thumb web.

Detaching flaps can usually be done in 14–20 days. The edge is usually sewed in accurately, but may be left open to be adjusted later. If this plan is followed, the detaching can be done simply under local anesthesia. Partial detaching can be done as indicated, severing part of the pedicle on two to three occasions. Delaying the base of the pedicle by making a regular delayed flat flap out of it is done, if there is a relative small attachment and if there has to be a large wrap-around or further let-in of the flap. This is carried out as a typical delayed flat flap and, of course, makes final detachment delayed.

The donor site can often be closed primarily or later, or may be left to heal. Grafting is done if it has not been done at the first operation or if too much more defect has been left when the arm is detached. There have been no gross complaints about the donor sites.

Double pedicles are occasionally used as are also pockets, and tubes.

Tubes, of course, are necessary for thumb reconstruction. But for the general mass of gunshot and shell fragment wounds the direct, nondelayed, nontubed flap is applicable.

Thinning flaps is necessary quite often. This fat is not a total detriment because it is this same fat that may make possible any deep work that is necessary. Flabby flaps on the palm, however, may prove to be about worthless for function.

Sensation develops surprisingly well in many flaps and, of course, depends on the presence of nerves in the area; if they are completely blown out, anesthesia will persist.

The following examples of the use of flaps are included as part of the text.

Figure 1 illustrates one of the most distinguishing features of war wounds —that is the lesions produced by shell fragmentation. The irregular, rough, sharp pieces of metal twist and swerve through tissue tearing up nerves, vessels and tendons and shattering bones.

The soft tissue of the palm and the thumb web has become board-like and the thumb fixed, and the roentgenograms show the reason for the loss of softness with the multiple fragments having been driven into the tissues.

Dissection of the scar is done through the whole area, and some of the fragments are shown on the gauze. The skin in this instance has to be taken to get rid of the deep scar.

A direct (or immediate, or nondelayed, or nontubed) flap is raised on the abdomen with the base up and from a thin-skin, hairless area.

The hand is planted in the flap, the arm and hand anchored with adhesive, and a cotton mechanics waste pressure dressing applied.

The hand is taken loose 16 days later and the edge is put down accurately. The abdomen is grafted at this stage, in this instance.

In this flap, fortunately, normal sensation developed and through it a secondary operation for suture of nerve slips to the fingers and for rotation of the thumb was done, with excellent function resulting.

Figure 2 illustrates a badly scarred forearm and the failure of a bone graft put in through dense scar—because the deep repairs can only be successful if the wound heals and does not break down.

Following removal of the bone graft (done elsewhere) the widespread scar was moved down to a good minute blood supply. A direct flap was designed and raised and the arm "planted" in it. Eighteen days later the flap was detached and wrapped further around the arm and sewed in place. The abdomen was grafted. A successful bone graft was put in through the flap and a satisfactory result obtained.

Figure 3 shows massive replacement of soft tissues of forearm in two operations with a direct flap, in 18 days' time. Abdomen grafted when flap was put on and closed when flap was detached.

This patient had asked that his arm be removed. Through this flap the wrist was fused and two tendon transplants were done to give a worth while result far superior to any prosthesis.

Figure 4 illustrates a direct dorsal flap that permits normal enough function so that the two procedures of dissecting the scar and putting the flap in place and detaching the flap 14 days later are all that is necessary.

Internal wiring to maintain separation of the thumb from the fingers during time of attachments to abdomen is shown in diagram.

Method of abbreviated recording, fingers, joints and bones by figures and letters is shown.

Conclusions are outlined in first paragraph.

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A DESIGN FOR SURGICAL CONVALESCENCE*

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Convalescence literally means *growing strong*. It may be defined as the period of recovery from the weakness produced by disease. In surgical convalescence, the disease is to a considerable extent an injury called the operation. Traditionally, it is assumed that convalescence does not really begin until after the effects of injury have ended; in other words, one cannot grow strong unless one has first grown weak. The processes of injury and healing are supposed to follow each other in chronologic order. Yet there is no real reason for believing that injury and repair must be consecutive rather than synchronous. This is of great practical importance, for if healing can begin before the injurious process is at an end, the length of convalescence can be considerably shortened and, perhaps, even made more effective.

Such a synchronous mechanism has been well described by W. G. MacCallum, who compared the process of injury and repair with a burning building. "Long before the fire is extinguished," he wrote, "workmen are found carrying away the charred timbers and enthusiastic carpenters are rebuilding wherever they can." If this opinion is physiologically sound, it is obvious that healing should be encouraged and stimulated as early as possible after the injury; in fact, almost at once. In the present study, therefore, convalescence will be considered as that period beginning immediately following operation.

A second traditional attitude toward convalescence puts great reliance upon the spontaneous efforts of nature. It gives special emphasis to rest. To be sure, active therapy after operation is now carried out, but it is usually confined to procedures combating the effects of trauma itself, such as the injection of saline and glucose, and perhaps blood or plasma transfusions, with the idea of lessening some of the deleterious effects of the injury. However, once these effects have come to an end, the process of healing is left strictly to nature. Even the patient's nutritional needs are left to the vagaries of his appetite.

Actually, there is no more reason why surgeons should rely upon the spontaneous efforts of nature during convalescence than at any other time

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unless such a passive policy really produces the best results. Medical history is replete with examples of scientific advances which developed because of impatience or disappointment with nature. While it is true that mortality from operations has steadily fallen, complications have gradually lessened, and the frontiers of surgery slowly extended, scientific advance should never be looked upon as having achieved its ultimate goal. Satisfaction often paralyzes, whereas dissatisfaction stimulates. Thus, a critical study of the spontaneous healing process called surgical convalescence is not only justified, but is most urgently needed to increase postoperative therapeutic possibilities.

Interest in surgical convalescence has been greatly stimulated by the present war, probably because the problems presented by large numbers of injured soldiers and sailors and marines accelerate clinical investigation. More important, however, is the fact that many of the injuries suffered in battle involve a long period of treatment, including the necessity for repeated surgical procedures. During this long hospital stay, many disabilities are observed which aggravate the problem of healing and prolong the convalescent period.

The term rehabilitation has been used to describe the later phases of convalescence. It implies special procedures for combating many of the deleterious effects of prolonged convalescence. From the physiologic point of view, rehabilitation and convalescence are intimately connected—the more efficient convalescence, the less need for rehabilitation. As a result, considerable study has been given to the process of healing immediately after disease and injury.

Much of this work has been initiated by the Army Air Forces, and a planned detailed convalescent program has been instituted with the aim of improving on the *laissez-faire* procedures of the past. Howard Rusk has reported that this plan of physical and educational training has reduced the length of hospitalization, served to maintain old or develop new skills, restored morale, and increased military knowledge.

Any information throwing light upon surgical convalescence may prove of considerable value not only in the care of soldiers injured in war, but also of those injured in civilian life, and even those injured by choice in a planned surgical procedure.

There is great danger that the lessons learned in wartime will be lost in peace. For example, a thorough convalescence program was worked out and used during World War I by John Bryant, who, with prophetic foresight, realized his work would "have been forgotten, to be rediscovered rather late in the next war." This was written in 1927. However, there is at present so much interest on the part of civilian surgeons in problems of convalescence that study will probably be continued after the war is over.

An important manifestation of this interest was the formation by Dr. L. H. Weed of the National Research Council in 1943 of a Committee on Convalescence and Rehabilitation. As a member of this Committee representing general surgery, the senior author was privileged to participate in

its meetings and activities. From the comembers and especially the chairman, Dr. Wm. S. Tillett, came much of the stimulus responsible for carrying out the present study. Many ideas expressed herein emerged from the group's deliberations, for which grateful acknowledgment is made.

FACTORS IN SURGICAL CONVALESCENCE

There are, of course, a great many factors which influence the healing process, including all the advances made in surgery during the past century. It is evident that excellent anesthesia and meticulous surgical technic, a careful aseptic ritual, chemotherapy, transfusions, *etc.*, are essentials without which the highest degree of healing cannot be achieved. These factors deal more directly with the operative procedure itself. They will not be discussed here, because their importance is realized and they have been given adequate attention elsewhere. In this study three nonsurgical factors will be described. These form a tripod upon which the present design for convalescence is based. They may briefly be described as follows:

A. *Psychogenic Factors.*—These deal with the personality of the patient and his reaction to the surgical procedure, both before and after the operation. They may exert either a favorable or a deleterious influence upon convalescence, depending to a considerable extent upon the surgeon in charge. Most of the deleterious influences are due to ignorance, fear and apprehension, based somewhat upon an accumulated background of misinformation sometimes transmitted by word of mouth or by misinterpretation of chance remarks overheard after entry into the hospital. Much of the influence of psychogenic factors has to do with the morale of the patient and with boredom, particularly when the surgical disease requires long periods of hospitalization and repeated surgical procedures.

B. *Physical Factors.*—These comprise changes due largely to the influence of bed rest and immobilization. The most important of these changes are those which lead to pulmonary and circulatory accidents. Less obvious and dramatic, though of considerable importance, are disturbances in neuromuscular abilities and the various changes described as the atrophy of disuse. The general term "deconditioning" is now often applied to the effects of immobility. Other physical factors associated with bed rest are more subjective and deal with the difficulty of performing such normal functions as defecation and urination in the horizontal position. Because all of these deleterious effects arise from immobility, it is obvious that they can be combated simply by encouraging movements from the very beginning of injury while the patient is still in bed, and by early termination of bed rest, technically called early ambulation.

C. *Nutritional Factors.*—These deal with the influence of operation on the nutrition of the patient; in other words, with the phenomenon of malnutrition, which practically always is seen after surgical procedures. Malnutrition is basically a wastage of body tissues, of which the most important is protein, and to a lesser extent loss of vitamins. In large part, this malnutri-

tion is due to starvation, *i.e.*, an inadequate dietary intake, based on anorexia, which is largely preventable. It also is due to the fact that surgical procedures lead to excessive losses, particularly of protein, much of which may be difficult to prevent or correct until after the effects of injury are over.

The three above-mentioned factors, while quite distinct, actually are intimately connected. For example, early termination of bed rest, when properly carried out, greatly improves the morale of the patient, eliminates many of his unfounded fears, and increases his appetite for food so that anorexia is automatically lessened. On the other hand, a good dietary intake increases physical strength and thus encourages an early return to full movement, which in turn improves appetite and morale and increases the capacity for physical activity. In this sense, there is a reciprocal influence of these factors one on the other. By taking advantage of all of them together, the beneficial effects obviously are increased.

PREVIOUS WORK

Convalescence, as such, has been the subject of surprisingly few publications. Up to 1938, one can find less than 150 titles in the entire bibliography published by the Surgeon General's Library under this general heading. Moreover, practically all of this literature is concerned with the period of convalescence following discharge from the hospital, with special reference to the establishment of so-called convalescent homes. As applied to a surgical patient, this has been called[1] the period "after the stitches were out and his hospital record neatly filed away."

A complete review of the literature on convalescence was published in 1927 in a monograph by Bryant, who also described his own experiences with a convalescence program he instituted in the medical corps of the United States Army during World War I. Included also was a chapter devoted to preoperative and postoperative care, titled the "Medical Aspects of Surgical Convalescence."

The most recent extensive publication on convalescent care followed a conference held in 1939 under the auspices of the New York Academy of Medicine.[7] All specialties were covered, including surgery, but the period discussed was that following discharge from the hospital, and dealt almost entirely with the advantages of special convalescent homes or hospitals. By contrast, the present study is devoted to the convalescent period preceding discharge and beginning immediately after the operation.

Consideration of the three factors already mentioned leads to further observations which have a bearing on the subject of convalescence. These will now be reviewed.

Psychogenic Factors.—The influence of psychogenic factors in surgery has received very limited study. Surgical diseases have such a large physical component that many surgeons would probably deny the influence of psychic factors entirely. Yet it is obvious that every individual reacts to psychic as well as to physical trauma in various ways, many of them deleterious,

and that these reactions are part of his clinical behavior even if he has a carcinoma of the stomach or a fracture of the femur. To overlook the influence of mind over body is to neglect an important part of the clinical picture and to miss significant therapeutic possibilities.

The work of George Crile on the deleterious effects of unpleasant and painful stimuli before and during operation falls into the category of psychogenic factors even though his studies dealt largely with physiologic effects, particularly on the circulation. Pharmacologic means to minimize preoperative apprehension are important and widely used. Excellent anesthesia in general and gentle operative technic also tend to minimize deleterious psychic trauma. While definite noxious stimuli, such as severe pain, are responsible for many of the psychic insults during and after operation, others are based on unfounded fears. Ralph Waldo Emerson realized this when he said—

> "Some of your hurts you have cured
> And the sharpest you still have survived,
> But what torments of grief you endured
> From evils which never arrived!"

A stimulating and direct approach to the study of psychogenic factors in surgical patients has been made by Barney Brooks. He brought a psychiatrist and a psychologist into the surgical wards for the study of patients, both before and after operation, by modern psychologic methods. The results, while only described in part, not only advanced knowledge of surgery, but also of psychiatry. It was interesting to note that only two patients on the ward failed to submit willingly and agreeably to such psychiatric investigation. On the other hand, private patients objected almost universally to such study. This difference was explained by the fact that the psychiatrists were not labeled as such on the surgical wards, but were permitted and even encouraged to pose as members of the surgical staff. Interestingly, preoperative apprehension was not common; many patients on the contrary really awaited operation as something to be achieved. Significant of the objective clinical value of the study were 14 consecutive cases of acute appendicitis, all showing acute inflammatory disease at operation; seven of these patients were found to have definite evidence of anxiety of sufficient gravity to be of clinical significance. In two other cases operated upon during the same period in which no organic change was found in the appendix, psychiatric examination disclosed sufficient evidence indicating that the clinical manifestations of acute appendicitis were really those of a neurosis.

In another clinic, a psychologic screening test was carried out in a large series of hospital patients by Wolff. The incidence of personality problems in general surgical as well as medical wards was found surprisingly high. At least 35 per cent of these patients exhibited changes of clinical significance, yet the attending physician and surgeon were unaware of the fact in any of them. In an extensive discussion prepared by the Committee on Convalescence and Rehabilitation of the National Research Council, many of these

psychogenic factors are described as they apply to all sorts of illnesses, including surgical cases.[31]

It is true, of course, that from time immemorial a surgeon with common sense and a real feeling for his patient automatically recognized the importance of psychogenic factors. Yet there has, in general, been an almost complete silence on the part of textbooks and teaching curricula regarding their influence. If such factors are of importance, they should be discussed and studied openly. If they are valuable during the surgical convalescence, they must be incorporated into any design for convalescence which has as its objective an improvement upon past efforts.

Physical Factors.—The virtue of rest as a therapeutic procedure is so deeply ingrained in the tradition of the healing art and has been so emphasized and extolled by the great leaders in medical thought for so long that it was almost considered heresy to describe and emphasize any deleterious effects which could be directly attributed to this general procedure. Yet as long ago as 1886 Lucas Championniere[22] denounced eloquently the evil effects of prolonged immobilization in the treatment of fractures. He expressed for the first time the belief that repair is more rapid and complete when some movement is permitted. He claimed that even pain is less with early movement because immobilization merely postponed pain which actually is worse the longer immobilization precedes the start of movement. He, therefore, advocated early mobilization and massage. It is noteworthy that he expressed the same idea 25 years later[23] with greater emphasis, based upon his added clinical experience.

Early mobilization of the entire body by shortening the period of bed rest after surgical operations was first carried out in 1899 by Emil Ries, a gynecologist practicing in Chicago. He described the beneficial effects of early ambulation in a series of patients who were allowed up on the first or second postoperative day following hysterectomy. This lead was not followed even by other gynecologists, for Howard A. Kelly, in 1911, wrote as follows: "The timid surgeon does not want to drag a miserable, depressed, suffering patient out on the floor so soon after an aggressive operation."

Yet the dangers of recumbency have long been realized by surgeons as far as older patients are concerned. Few surgeons would hesitate to get a patient out of bed almost immediately after operation if the patient were in his 80's or 90's. The advantages of such early termination of bed rest are freely acknowledged. The deleterious effect of prolonged bed rest in the aged has been realized even in other types of disease, the evidence being summarized very well by LaPlace and Nicholson in 1938. Although early ambulation produced no deleterious results in the aged, it was not widely used in younger patients. Most textbooks recommend, as a routine, bed rest for at least ten days following abdominal operations.[9]

A complete collective review of observations on early termination of bed rest after operation was published in 1943 by Newberger.[28] In this review only 44 of the 189 references to the literature were in the English language and

nearly all were published previous to 1914. With few exceptions, and those from European centers, the communications were all from nonuniversity clinics. By 1922, two English surgeons, Claremont and Rowland, stated the case for early termination of bed rest convincingly. As a result of Newberger's review, one definitely gets the impression that early ambulation has many advantages, without real disadvantages, and one wonders why the idea failed to gain general acceptance. Perhaps it was due to a lack of confidence in the security of catgut sutures with which most wounds were closed. Experimental observations have been very meager indeed, but Newberger himself[29] carried out a study on the tensile strength of healing wounds in rats, and was able to show that immobilized animals exhibited a definite difference as compared with those who are exercised. While the tensile strength in the two groups was the same on the third and on the tenth days, the exercised animals showed a definitely greater tensile strength at the fifth day.

"The Abuse of Rest in the Treatment of Disease" formed the subject of a symposium held in 1944 at a meeting of the American Medical Association. Medical, surgical and other types of disease were discussed.[36] A paper by Powers presented detailed data in regard to the effect of early ambulation after a variety of abdominal operations as compared with control cases treated by traditional methods. The evidence was clearly in favor of early ambulation.

Two other communications on the early termination of postoperative bed rest have appeared in the past few years. Leithauser, in 1943, as a result of his experience, was very much in favor of early ambulation. He included observations on the vital capacity of 21 of his cases, 13 appendicectomies and eight cholecystectomies. The return to a normal vital capacity was much more rapid as compared with measurements reported by others in patients treated by the usual period of bed rest. Nelson, on the basis of over 400 personally observed cases, was also greatly impressed with the value of early postoperative ambulation. Only three cases exhibited partial wound disruption and only two developed small incisional hernias. In the present war a number of studies have been made on the beneficial effect of early ambulation following abdominal operations, and especially in head injuries in military personnel. A witty and provocative editorial in a recent number of the Lancet[12] entitled "Keep Moving Please" summarized much of the current English reaction to early ambulation after operation.

Among the evil sequelae of complete bed rest, interestingly discussed by Dock, are circulatory changes associated with thromboses and emboli. There is, of course, a large literature on the etiology of postoperative thrombosis and embolism. The influence of bed rest and immobility is convincingly shown in a recent study by Hunter, Krygier, Kennedy and Sneeden. They dissected the leg veins in 169 cases at autopsy for the presence or absence of thrombosis. All cases had been in the hospital for 48 hours or more before death. They were divided into two groups: (1) Those who exercised in bed and were ambulatory up to 48 hours before death; and (2) those who

did not exercise and were nonambulatory. In the first, or ambulatory group, only five medical and two surgical cases showed thrombosis, an incidence of 17.9 per cent; in the second, or nonambulatory group, 42 medical and 27 surgical cases showed thrombosis, an incidence of 53.1 per cent. In a discussion of the literature on the etiology of thrombosis, these authors emphasize confinement in bed as the common denominator of all studies.

Other effects of bed rest could be discussed. Fairly well known are all the changes associated with the atrophy of disuse. These occur to a lesser or greater degree depending on the degree and duration of immobility. Then there are metabolic changes, alterations in cardiovascular mechanisms, loss of neuromuscular skill, *etc.* Many of these changes have been discussed in a "Symposium on Physiological Aspects of Convalescence and Rehabilitation" published by the American Physiological Society.[37]

It is obvious from this brief résumé of the literature that bed rest and immobilization cannot be viewed with complacency. Their effects must be looked upon objectively, and their disadvantages weighed impartially against their advantages with accurate attention to detail in terms of the actual results achieved. Bed rest and immobilization have well known and definite beneficial effects which must be utilized without, if possible, producing any deleterious results. To do so completely, probably is impossible.

The elusive quality of judgment must be used in order to decide just where to draw the line in regard to the termination of bed rest. Certainly, it should be utilized only insofar as it achieves beneficial results and no longer. When its advantages begin to diminish, it should be terminated at the point where the advantages of mobilization more than outweigh the disadvantages of immobilization.

From the studies reported, it would seem that early ambulation has the following advantages: a lowered incidence of postoperative, particularly pulmonary and vascular, complications; less nausea, vomiting and abdominal distention; an earlier return of normal functions of the bladder and the bowel; a beneficial psychologic effect on the patient's morale and mental status; and the acceleration of convalescence permitting an earlier return to working ability with resultant economic savings to the patient.

Nutritional Factors.—In view of the wide interest in nutrition, it is surprising to note the dearth of study of the general nutritional changes after surgical operations. Yet it is a commonplace observation that practically every surgical patient suffers some loss of body weight. This has been considered inevitable and, for this reason, was perhaps overlooked. A brief discussion of the nutritional factors in convalescence was written by the Committee on Convalescence and Rehabilitation of the National Research Council.[26]

Wastage of body tissue in itself may be of little physiologic significance unless protein tissue is affected. Weech has shown that plasma proteins begin to fall with the very onset of a protein-free diet. That protein frequently is lost in surgical patients is shown by the high incidence of postoperative hypoproteinemia, a defect which often has serious clinical implications. Attention

was first called to this deficiency, particularly as a cause of postoperative edema, in 1933, by Jones and Eaton. Since then, postoperative protein deficiencies have been emphasized by many observers.[13]

Of the causes of malnutrition in surgical patients, the most obvious is an inadequate dietary intake. This is due to one or more of several factors.[14] If the patient is able to eat, starvation occurs either because a restricted diet is imposed upon him, or because of anorexia which may follow surgical procedures. If the patient is unable to take anything by mouth, starvation is due to the fact that, although the parenteral route is used, the fluid given contains inadequate calories, vitamins and usually no protein.

A second mechanism often responsible for malnutrition is the excessive loss, particularly of protein, which occurs as a result of the operative procedure. This excessive loss occurs in two ways. The first is actual spilling of blood itself or extravasation of blood, plasma or exudate into the traumatized area, thus, resulting in hypoproteinemia, or anemia, or both. The second is a breakdown of protein tissue, occurring after operation and in many infections, which has been described as "the toxic destruction of protein." The magnitude of these losses may be tremendous; whereas the normal requirement for protein balance usually is placed at about 70 Gm. per day, the amount lost after operation may reach a figure which is two to four times as great. In other words, to prevent any protein starvation, a postoperative patient might have to assimilate the equivalent of 200 to 300 Gm. of protein each day. This actually has been done by increasing the intake of protein to this level.[8] Increased physical strength, weight gain and more rapid convalescence were observed. On the other hand, other observations have indicated that, in well nourished patients, protein losses for a certain period after operation cannot be corrected because nitrogenous food given during this catabolic phase cannot be assimilated.[3]

Clinical manifestations attributable to protein starvation after operation have been summarized in a previous communication.[14] They include hypoproteinemia, postoperative weakness, asthenia and anorexia, nutritional edema, lowered resistance to infection, impaired liver function and, in extreme cases, a fatal outcome. There is a growing realization that starvation cannot be disregarded because many postoperative complications are directly attributable to it. Starvation is no longer necessary even if the patient can take nothing by mouth; methods are now available for almost complete parenteral alimentation.[16] Besides protein, postoperative deficiencies in vitamin C have been observed in normally nourished patients as part of the operative trauma.[24] Whether thiamine or other vitamins are lost or destroyed after surgical procedures is not known.

THE PRESENT DESIGN FOR CONVALESCENCE

The design for convalescence used in the present study was based on the psychogenic, physical and nutritional factors already discussed. The influences of these three factors were studied in each of 79 consecutive surgical

cases. All patients admitted to one surgical female ward were observed for a period of four months, from April to September, 1944, and all male patients admitted to another ward for a succeeding four months, from September, 1944, to January, 1945. As controls, the same wards were used on the alternate periods, the patients receiving during this time the customary postoperative regimen. This admittedly is not an ideal method of clinical study, for it would have been preferable to study concurrent controls selected alternately as they were admitted to the ward.

Unexpectedly, we soon observed that the establishment of this design on all patients of a particular ward in itself had an interesting result—rivalry between patients in their desire to achieve complete recovery. They encouraged each other to carry out the program of early movement and to increase their dietary intake—about which they were all told ahead of time. It was clear that the morale of the ward was definitely improved and that, inadvertently, we were achieving what is often called group psychotherapy.

The details of the program were carried out as follows: Each patient on admission had an interview with the visiting or the resident surgeon who discussed completely the plans for diagnosis and for treatment. The objective to be reached was described and emphasized. Each patient was told that we expected and would encourage him to walk within a few days after operation, but that he would not be urged to do so unless he wished. Fear was usually the greatest deterring factor. Patients were told that their wounds would be so securely sutured that there was no danger of disruption, and they need fear no accidents by following the program. Once they heard of other patients walking within a few days after operation or actually saw this themselves, their fear nearly always disappeared. For example, one of our patients, a 66-year-old male, following gastric resection, elected to start walking on his third day. When his friend in the next bed saw this, he decided to do even better after his gastrectomy. He got up and walked on his second postoperative day, in spite of his 72 years of age and an ankylosed hip. Each patient was told that feeding would be given intravenously while nauseated, but that he would be expected to eat within a few days after operation, at first liquids, but soon solid food. They were told that walking would be encouraged in increasing lengths each day, and that we fully expected them to leave the hospital feeling as strong or even stronger than they did when they entered. They were then questioned as to the existence of any fears. They were encouraged to tell of any disturbing experiences that friends of theirs had had following operation, and the facts were explained. Their own plans after leaving the hospital were discussed when possible or necessary. In discussing these matters, each patient was considered individually. A rigid routine was avoided as much as possible; only the general outlines of the design were followed. In patients depleted by their disease, preoperative preparations included procedures such as correction of nutritional deficiencies by various methods not germane to the present discussion.

Immediately after the more serious operations, an attempt was made to

achieve complete replacement therapy. A sufficient transfusion of whole blood and/or plasma was given to correct any losses resulting from the operation itself. The amount varied from 500 to 2,000 cc. of whole blood and from 500 cc. of plasma to 1,000 cc. Postoperative hematocrits and plasma protein determinations helped to decide whether deficits still existed even after such replacement therapy. A general anesthetic was employed in nearly all cases. Nonabsorbable sutures of silk and stainless steel were used.

As soon as the patient was conscious and rational after awakening from the anesthetic, he was reminded that he was to move about in bed at regular intervals. Pain, of course, was the usual deterrent, but sedatives were used only when necessary. Physical methods and suggestion, such as the achievement of a comfortable position and reassurance, were preferred. There usually is a good deal of pain in the wound for 24 hours, but much of it may be avoided if the skin sutures have been placed loosely. Early movement of the patient was generally confined to regular exercising of the legs and of the arms and periodic overventilation by deep breathing. The patient was encouraged to turn frequently. Almost immediately he was allowed to sit up in bed at intervals. Each day the question of walking was discussed with the patient. In any case, he was permitted to sit on the edge of the bed with his feet dangling the day after operation. In most cases, patients could be encouraged to stand on their feet by the second or third day. In only a few cases did this occur after the fifth day. In Table I these figures are listed, together with the various operations carried out.

Nutritional requirements were met at least in part from the very first. If the patient could eat he was encouraged to do so at once. In most cases vomiting will follow attempts to drink even water, particularly following a general anesthesia; in other cases the oral channel cannot be used because the surgeon wishes to keep the gastro-intestinal tract at rest. In either case the parenteral route must be used.

The usual routine after the more severe operations was the intravenous injection of two liters containing 100 Gm. each of glucose and hydrolyzed protein (amino-acids)* and about 5 Gm. of sodium chloride at a p_H of 6.5. One liter was usually given in the morning, and one in the afternoon. Often an additional liter was given of 5 per cent glucose in water (or saline if indicated). One gram of vitamin C was injected into the muscle each day. Two other daily injections were given containing thiamine, riboflavin and niacin. This regimen was used for as short a period as possible, *i.e.*, until sufficient food was taken by mouth. Patients were encouraged to take fluids by mouth as soon as the surgeon considered it safe. This often occurred within a day or two in simple cases, or as soon as the effects of the anesthesia had worn off. In the case of gastric resections the period was longer, often three or four days, before liquid was permitted. At first simple fluids such as tea, broth and fruit juices are given, but if tolerated without vomiting or distress, milk is added. A high protein drink consisting of skimmed milk

* Amigen.

powder added to milk, 100 Gm. to the glass, is often a useful way of increasing the protein intake.

The importance of giving priority to protein is suggested by experimental studies[15] showing that in the absence of malnutrition much of the caloric requirement may safely be met by the patient's own tissue fat. But even liquids are supplemented within a day or two by eggs and other solid foods which are added as soon as possible. In all cases except gastric resection a full, well balanced diet was taken by the end of the first week following operation. Considerable individual variation was observed. This often required special attention to idiosyncrasies of the patient's appetite when it did not interfere with the ingestion of good food.

No specific attempt was made to increase the morale of the patients or to combat boredom in any special way. Most civilian patients are in the hospital for such a short period that factors of morale probably are not as important as they are for military personnel, especially when prolonged hospitalization is necessary. Nevertheless, we are convinced that an active program of education, diversion and entertainment has its place even in an ordinary surgical ward and particularly when the patient is in a single room and when hospitalization is prolonged. Such measures, including occupational therapy, will beneficially influence surgical convalescence.

In a number of patients, significant chemical data were obtained. The 24-hour urinary output was collected and its nitrogen content determined. Specimens of heparinized blood were collected and cell volume, plasma albumin and globulin determinations carried out. These patients were weighed before operation and on discharge. Chemical methods for fractional plasma proteins were those described by Campbell and Hanna. The Kjeldahl procedure was that of Sobel, Yuska and Cohen.

PRESENT FINDINGS

The results of this study may be listed under the headings of (a) bedside or clinical observations; and (b) chemical and physical or laboratory measurements.

Clinically, the bedside findings may be described, first, in terms of beneficial effects and, second, in the absence of deleterious ones. In regard to the first, the beneficial clinical observations were quite evident in the general condition of these patients. They felt better and exhibited much less of the postoperative asthenia and depression which may follow a serious abdominal procedure. This was evident both to the attending physician and the nurses in attendance on the ward. There was an obvious decrease in the amount of nursing and attendant care, particularly in the serving of trays and the routine making up of beds, especially in the female ward, because the patients were able to take care of themselves early in their postoperative course.

The most striking observation, however, was the clinical condition of the patient at the time of discharge, which, in this series, was not reduced in length from the usual fortnight. Invariably, they felt quite strong and vig-

orous. Many of them voluntarily expressed their ability to do a good day's work. Indeed, many patients after early ambulation stated that they were strong enough to leave the hospital and to go home even before the stitches were removed. They often objected to our requirement that they remain the usual fortnight. This was in striking contrast to the asthenic condition of the control patients who were often permitted to go home the next day after they got out of bed. This clinical result was well expressed by two private patients treated by the present design. The one, after a cholecystectomy, stated that she had made plans to spend six weeks with her mother recuperating. On discharge she felt no need at all for such recuperation, yet she had not made plans to return to work. She was very much pleased when it was suggested that she enjoy an active vacation. The other, after a partial gastrectomy, left the hospital on his 12th postoperative day and called the next day requesting permission to return to his desk job.

On the negative side, regarding the diminution of deleterious effects, there was no clinical evidence of embolism in any of the 79 cases. During this same eight-month period, among the control cases treated on the same wards, three vascular accidents were observed among patients who were not treated by the present design and who were kept in bed for the usual ten-day period. In two of these patients, the accident proved fatal. One of them was a patient who had had a saphenous ligation. Because the wound became infected, she was kept in bed for seven days. Death occurred suddenly as soon as she was allowed to stand. The second case was an uneventful cholecystectomy who was kept in bed for ten days and in whom sudden death followed immediately her getting out of bed. The third was an ileostomy for ulcerative colitis, who on the 12th day, while still in bed, developed severe evidence of pulmonary thrombosis from which, however, the patient gradually recovered.

In regard to wound disruption, there were none in the entire series of 79 cases. In the control group of patients, two wound disruptions were observed, both requiring an anesthesia and resuture. No observations were made as to the degree of postoperative fever, nausea or vomiting, but the clinical impression was gained that these manifestations were certainly no more pronounced than usual, indeed, probably less so.

The more objective measurements consisted of studies on nitrogen balance and in measurements of the serum proteins and changes in the body weight. Only the data which are more or less complete are presented; these are shown in the accompanying charts. It will be observed that in the control cases there was a loss of weight of about eight pounds and an average fall in the plasma albumin of .20 Gm. per cent between the preoperative measurement and that on discharge as compared with a loss of but one pound and a gain of .15 Gm. per cent in the plasma albumin in the group treated by the present design. (See Tables II and IIa.)

Comparison of the nitrogen losses in the urine shows a somewhat greater depletion in the patients treated by the traditional routine. This is seen

readily by comparing the losses in the first three days, a period during which no nitrogenous food was taken by mouth in either group. The average loss in the control group receiving no nitrogenous food was 5.8, 8.6 and 9.2 Gm. for these three days, even though the patients had much less serious operations than the group in whom the present design for convalescence was carried out. In this latter group the urinary excretion of nitrogen in the first three days after operation was much greater, yet the negative nitrogen balance was much less, *i.e.*, 4.0, 2.7 and 3.9 Gm. for these three days. This was due to the intake of nitrogen as hydrolyzed protein. (See Tables III and IIIa.)

TABLE I

SUMMARY OF THE OPERATIONS PERFORMED IN THE 79 CASES STUDIED, AND THE POSTOPERATIVE DAY WALKING WAS STARTED

	Days after Operation when Complete Bed Rest was Terminated							
Operation	1	2	3	4	5	6	7	Total
Inguinal herniotomy	12	4	1	4	1	1		23
Appendicectomy	2	7	3	2				14
Ventral herniotomy, with fascia transplant	1	2	1	1	1	1		7
Cholecystectomy		2	2	1				5
Gastric resection		2	4	1				7
Resection of rectum (abdomino-perineal)		1	1		2	2	1	7
Resection of colon	1		1	2				4
Splenectomy		3						3
Celiotomy & misc.	4		4	1				9
Total	20	21	17	12	4	4	1	79

TABLE II

CHANGES IN BODY WEIGHT AND PLASMA ALBUMIN IN PATIENTS RECEIVING TRADITIONAL POSTOPERATIVE CARE

				Plasma Albumin (Gm. %)			
Patient	Sex	Age	Operation	Before Operation	Change at Discharge	Change in Body Weight (Lbs.)	Days between Operation and Discharge
1. B. E.	F	26	Inguinal herniotomy	4.49	+ .23	— 4	10
2. C. D.	M	59	Appendicectomy	4.83	+ .04	— 3	20
3. H. S.	M	55	Appendicectomy	4.69	— .42	— 8	11
4. A. S.	M	68	Appendicectomy	4.27	+ .19	—10	20
5. M. Y.	F	39	Appendicectomy	4.41	— .13	— 9	18
6. A. P.	F	63	Ventral herniotomy	4.19	— .13	—12	36
7. M. B.	F	50	Appendicectomy and cholecystectomy	3.86	+.16	— 3	13
8. S. L.	M	55	Gastric resection	4.61	— .32	—10	12
9. L. H.	F	47	Abdomino-perineal resection of rectum	4.23	— .10	— 9	26
10. F. R.	F	46	Resection of colon	4.04	—1.55	—17	36
11. B. R.	F	31	Cholecystectomy	4.56	— .20	— 5	12
12. I. B.	F	65	Cholecystectomy	4.19	— .22	— 6	12
Average				4.36	— .20	— 8	19

Note that there was an average fall of .20 grams per cent in the plasma albumin level. Note also that there was a loss of weight in each case even following the simple procedure of herniotomy or appendicectomy, the average loss being eight pounds.

These patients were given saline and glucose after operation until they were all able to take simple fluids by mouth. A gradually increasing diet was then permitted. Bed rest lasted for at least eight days following operation.

The above observations are to be compared with a concurrent group of patients (Table IIa) receiving the postoperative care described in this paper.

During this study it seemed apparent to us that patients given parenteral protein and vitamins from the start of the postoperative period began to eat more and sooner and felt stronger than the control cases receiving glucose

and saline alone. In many cases anorexia was apparently based to a considerable extent upon protein starvation itself. This statement may be surprising to many. It may even be questioned by some because it is usually assumed that the patient's appetite can be relied upon to determine when and how much food he receives.

TABLE IIa

CHANGES IN BODY WEIGHT AND PLASMA ALBUMIN IN PATIENTS RECEIVING THE POSTOPERATIVE CARE DESCRIBED HEREIN

Patient	Sex	Age	Operation	Plasma Albumin (Gm. %) Before Operation	Plasma Albumin (Gm. %) Change at Discharge	Change in Body Weight (Lbs.)	Days between Operation and Discharge
1. L. H.	F	41	Cholecystectomy	4.40	+.71	—1	11
2. T. R.	F	50	Cholecystectomy	4.43	—.14	+3	12
3. A. W.	F	64	Abdomino perineal resection of rectum	4.50	—.37	..	18
4. P. B.	F	42	Abdomino-perineal resection of rectum	4.28	+.35	—2	21
5. E. B.	F	55	Abdomino-perineal resection of rectum	3.95	+.19	—1	26
6. H. C.	F	62	Abdomino-perineal resection of rectum	3.79	+.75	0	47
7. M. W.	F	57	Resection of colon	4.01	—.01	—4	17
8. A. W.	F	61	Resection of colon	4.27	+.27	0	14
9. M. C.	F	25	Resection of colon	3.64	+.76	+3	17
10. B. H.	F	27	Ventral herniotomy	4.63	+.08	—3	12
11. J. Z.	M	66	Gastric resection	4.38	—.47	—1	18
12. S. S.	M	36	Gastric resection	4.26	—.30	—8	15
Average				4.31	+.15	—1	19

Note that there was a slight increase in the average plasma albumin, even though the initial value before operation was similar to the control group listed in Table II. Note also an average loss of only one pound in body weight at the time of discharge, even though the operations in the above cases were much more serious than in the control group.

These patients were all treated according to the design for convalescence described herein.

TABLE III

TOTAL URINARY NITROGEN EXCRETED BY PATIENTS NOT RECEIVING HYDROLYZED PROTEIN (AMINO-ACIDS) BY VEIN AFTER OPERATION

Patient	Sex	Age	Operation	Postoperative Days 1	2	3	4	5	6
1. H. L.	F	40	Cholecystectomy	...	12.5	13.5	9.6	7.9	10.6
2. M. F.	F	46	Ventral herniotomy, with fascial transplant	...	8.5	8.4	...	7.5	8.5
3. G. W.	F	39	Ventral herniotomy, with fascial transplant	4.3	9.3	9.0	8.5	8.4	7.0
4. J. G.	F	34	Ventral herniotomy, with fascial transplant	5.5	5.5	7.4	8.6	12.6	9.0
5. J. B.	F	25	Splenectomy	...	3.9	...	12.0	9.7	8.2
6. B. B.	F	17	Splenectomy	5.0	10.1	7.9	13.1	9.1	7.1
7. M. H.	F	30	Splenectomy	...	12.4	11.1	10.9	9.0	10.7
8. E. W.	F	54	Removal ovarian tumor	10.5	...	7.7	9.8	7.5	7.4
9. J. F.	F	55	Radical mammectomy	4.0	6.6	9.2	9.7	7.7	...
Average				5.8	8.6	9.2	10.2	8.8	8.6

Note the absence of any large excretion of nitrogen, the highest being 13.1 grams. The figures for the first three days represent negative nitrogen balance because intake during this period was confined to injections of glucose and saline solutions and simple liquids by mouth such as tea and fruit juice. Thereafter an increasingly full diet was taken so that the negative balance would be somewhat less. How much less cannot be stated, inasmuch as measurements of the nitrogen intake were not made.

While appetite probably is an adequate guide in a good many young healthy individuals after simple operations, it is apparently not so after more severe procedures, in those already malnourished at the time of opera-

tion, and particularly when fear of eating becomes superimposed upon the natural craving for food. The development of thiamine deficiency, particularly after intravenous injections of glucose, also may play a part in many cases of postoperative anorexia. Even in normal individuals, a voluntary fast will often dull the sensation of appetite after the first or second day. This undoubtedly occurs in surgical patients deprived of protein for a few days after operation. Thus, malnutrition persists and with it comes a

TABLE IIIa

TOTAL URINARY NITROGEN EXCRETED BY PATIENTS RECEIVING HYDROLYZED PROTEIN (AMINO-ACIDS) BY VEIN AFTER OPERATION

Postoperative Days				1		2		3		4		5	
Patient	Sex	Age	Operation		B		B		B		B		B
1. L. H.	F	41	Cholecystectomy.. . .	5.6	+6.4	14.3	—2.3	17.9	—5.9	9.5		6.2	
2. T. R.	F	50	Cholecystectomy.....	13.7	—1.7	10.8	—4.8						
3. V. S.	F	28	Cholecystectomy.....	5.7	+.3	5.6	+.4						
4. M. M.	F	55	Cholecystectomy.....	—	—	15.5	—3.5	10.9	—4.9				
5. A. W.	F	64	Abdomino-perineal resection of rectum....	—	—	13.1	—1.1						
6 P. B.	F	42	Abdomino-perineal resection of rectum....	9.3	—3.3	12.1	—.1	14.1	—5.1	14.0	—5.0		
7. E. B.	F	55	Abdomino-perineal resection of rectum....	19.1	—13.1	14.2	—2.2	16.4	—4.4	17.1	—5.1	9.2	—3.2
8. H. C.	F	62	Abdomino-perineal resection of rectum....	12.6	—6.6	14.3	—2.3	17.3	—5.3	13.5	—1.5	13.5	—1.5
9. M. W.	F	57	Resection of colon.....			17.0	—5.0	22.2	—4.2	15.6	—3.6	8.2	+3.8
10. A. W.	F	61	Resection of colon....			14.1	—2.1	14.4	—2.4				
11. M. C.	F	25	Resection of colon....	6.6	—.6			9.2	+2.8	11.8	+.2	12.7	—.7
12. B. H.	F	27	Ventral herniotomy...	10.8	—4.8	11.1	+.9	13.5	—1.5	9.1	+2.9		
13. W. W.	M	70	Gastric resection......	7.1	—1.1	11.6	—5.6	13.4	—1.4	15.1	—3.1		
14. A. A.	M	30	Gastric resection......					29.6	—17.6	23.3	—11.3	18.7	—6.7
15. J. Z.	M	66	Gastric resection......			8.8	+3.2	11.6	+.4	15.5	—3.5		
16. L. L.	M	42	Gastric resection......	11.4	—11.4	21.1	—9.1	12.9	—.9	13.2	—1.2		
17. S. S.	M	36	Gastric resection......	10.3	—10.3	20.0	—8.0	23.2	—11.2	20.0	—8.0	17.8	—5.8
18. I. B.	F	28	Closure of fistula.....	2.2	—2.2	8.6	—2.6	6.9	+2.1	15.1	—3.1	17.4	—5.4
Average nitrogen output..................				9.6		13.2		15.5		14.8		13.9	
Average nitrogen balance..................					—4.0		—2.7		—3.9		—3.7		—2.7

Note the absence of any large excretion of nitrogen except in Cases 14 and 17, in which severe pulmonary atalectasis developed.

Under column B are listed the daily nitrogen balances obtained by subtracting the nitrogen output from the nitrogen in the intravenously injected hydrolyzed protein (amounting usually to 12 Gm. per day). Note that while positive balance was not often achieved, the degree of negative balance was considerably lower than in the group listed in Table III in cases *not* receiving parenteral protein feeding.

The nitrogen injected in the form of blood and plasma transfusions was not included in the above figures nor in those of Table III.

further impairment of appetite. This may lead to a vicious circle. Many surgical patients undoubtedly reach a state of severe malnutrition in this way, particularly when bed rest and immobilization have formed part of the postoperative regimen, and when the disease itself has increased the requirements for food. These observations would seem to emphasize the advantage of avoiding even short periods of starvation after operation.

SUMMARY

A series of 79 patients was studied after a variety of abdominal operations, including some serious ones. Special attention was paid to elimination of deleterious psychogenic factors before operation and during the convalescent period. Movement in bed was begun immediately, and bed rest

was terminated within a few days after operation. Complete starvation was avoided by the inclusion of hydrolyzed protein (amino-acids) in the parenteral injections of glucose and vitamins from the very beginning. Early resumption of a normal oral intake was achieved.

This design for surgical convalescence seemed to produce definite beneficial results when compared with the results in the control group of patients subjected to traditional postoperative regimen. These results were both clinical and chemical. The former included a more or less complete rehabilitation of the patient by the time of discharge from the hospital, thus, eliminating the necessity of further convalescent care. The latter included less loss of weight and an increase rather than a decrease in the concentration of plasma albumin over the control cases. There was a lower negative nitrogen balance in the cases given hydrolyzed protein (amino-acids).

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PROFITS TO PEACE-TIME PRACTICE FROM SURGICAL EXPERIENCES OF WAR*

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THE SURGICAL LESSONS of war are learned in a hard school. Their application is immediate and directed to the business in hand. They are learned in the laboratory of day-by-day experience. It is a school which has gathered in its faculty the great teachers and research students of the time; yet it is a school in which every student must be a teacher and every teacher becomes, in turn, a student.

There is no vacation or recess in this surgical school of war, it will remain in session as long as the fighting lasts and until the last wounded soldier has attained "maximum hospital benefit." Yet, as combat draws to a close there will be an increasing number of graduates from this school. From battlefields and hospitals they will return to the less exacting curriculum of peace. Their diplomas will read simply: "Honorably Discharged," and carry no degree. As alumni of this school, they will carry with them to their offices, and clinics and hospitals a certain fund of dearly-bought knowledge.

Perhaps, in its somewhat limited field of application to civil practice, this knowledge may not be worth what it has cost in time and effort and lives to acquire. What has been gained is but of the greater value because of its price.

It may be well to anticipate this commencement a little, to count the gains that have been made and the progress that has been achieved in surgical practice and investigation under the urgent stimulus of war. What will the graduating Army surgeon carry with him, as a result of his experience, that will help and guide him in the pursuit of peace-time practice?

Certainly, the surgical experiences of war will have their most direct bearing upon the less common injuries of peace. The industrial accident, the automobile crash, the train or plane wreck or the disastrous fire will provide an occasional victim upon whom the war-time arts of surgery may be beneficently practiced. Fortunately these will be relatively few, but there is no doubt that the knowledge required for their better treatment has been not only increased, but much more widely disseminated because of the war.

Emergency Treatment.—In dealing with such emergencies, though our facilities in peaceful communities are more stable and readily at hand, we shall not forget the lesson of the need for rapid and safe transportation of the patient to them for the earliest possible skillful care. We know the necessity for aseptic protection of the wound and of strict immobilization of the injured part. We know the life-giving values of fluids administered intravenously, of plasma, of whole blood, either fresh or stored, and of serum albumin and of the need for their quick availability.[1] We have learned, un-

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forgettably, the necessity for the intelligent employment of these materials and know how, by simple laboratory methods, to evaluate quickly and accurately the patient's need of them. Thanks to war-time experience the significance of blood count, specific gravity and hematocrit and the clinical import of their relationship are matters of common knowledge.

The emergency treatment of wounds has come to be much better understood. The value of protection from secondary infection and the deleterious effect of motion in transport of even soft-tissue injuries have come better to be recognized. The advantages of thorough yet tissue-conserving débridement, learned in World War I, and partly forgotten, have received fresh emphasis.[2] The menace of dead tissue and respect for living tissue is a lesson every Army surgeon has learned. There has been gained a new concept of traumatic vasospasm, resulting from vascular, nerve and tissue injury, as a danger to the circulation of the compromised extremity. The values of débridement, sympathectomy and sympathetic block in combating this condition have been firmly established.

The use of plaster casings, after a period of overemphasis, as a means of definitive treatment in open wounds, has assumed its proper place as a measure of protection during transportation. Burns have come to be properly regarded as infectable surgical wounds entitled to full aseptic respect. Largely as a result of war experience the damaging effect of escharotics and the value of pressure dressings and noninterference have been recognized.[2] The advantage of the promotion of early healing, in these and all other large open wounds, by prompt skin-grafting has been well learned. In major injuries of the extremities the Army has taught when, how and where to amputate.[3] The principles it has laid down and consistently followed will unquestionably result in conservation of life and limb and the better fitting of prosthetic appliances in future civilian traumatic practice.[4]

Bacteriostatic Drugs.—The need for combating and controlling infection in large numbers of wounded has provided an opportunity for clinical experience with bacteriostatic drugs that could not have been equalled in many years of civil practice. The knowledge thus gained will surely be employed to the future benefit of patients everywhere. Intensive experience with these drugs in military practice has given a new concept of the surgery of infection. The brilliant promise of the sulfonamides has been exceeded by the performance of penicillin.

It has been amply demonstrated that the prompt and continued administration of sulfadiazine upon the receipt of injury is a valuable prophylactic measure against surgical sepsis. Less certain, and open to some question, is the value of the topical application of sulfa drugs to injured or infected tissue; but the weight of evidence indicates the greater efficacy of their systemic administration. Some of the dangers and limitations of sulfa drug therapy have also been recognized, and precautions in their use have been established. Individual drug sensitivity and reactions have come to be better recognized, and urinary lithiasis, damage to renal tissue, and to the hemo-

poietic system are watched for and guarded against.[5] The natural resistance of certain organisms to sulfa medication has been quite firmly established, and the fact of the ability of some bacteria to acquire such resistance has been learned. These factors have modified considerably the use of these drugs.

Penicillin, used for a time almost exclusively by the military, has been put to an extended and searching clinical test. The results will greatly influence future surgical practice. Its brilliant effect in forestalling the activity of invading micro-organisms has taught the possibility of delayed primary closure of débrided grossly contaminated wounds. The converting of destructive compound fractures into clean simple fractures within a few days under its systemic administration is but one example of its efficacy. Its effect on infection already established is hardly less striking. The control it exerts in osteomyelitis and prolonged suppuration, the rapid subsidence of local infections of boils and carbuncles, of the tissue spaces of the hand and foot, of bones and joints, of empyema and lung abscess, and in contaminated operative wounds have taught an entirely new concept of the surgical management of such conditions.[6]

Yet war-time experience with the bacteriostatic drugs, because of its intensity and extent, has not permitted the development of an attitude of over-enthusiastic acceptance of their benefits. The experience has given opportunity for a more complete and just evaluation of their usefulness and shortcomings. It has been shown that their effects on certain organisms, notably of the *Escherichia* and *Clostridia* type, is negligible, and that even organisms susceptible to their effects may become resistant in time. The lesson has been firmly impressed upon the surgeon that the sulfas and penicillin are tools of extraordinary value and usefulness which will greatly supplement but cannot supplant those therapeutic procedures that are based on sound surgical principles. The complete removal of foreign matter and devitalized tissue, protection and the maintenance of strict asepsis are still the keystones of successful wound surgery.[2]

Fractures and Bone Surgery.—The management of fractures, simple as well as compound, occurring in great numbers in war-time, has provided an intensive experience for the Army surgeon. This experience has taught him the value and applicability of accepted treatment methods and has furnished an opportunity for their comparison and evaluation. As a result the advantages of judicious use of skeletal traction-suspension methods of the secondary treatment of fractures, especially of the major long bones, have been confirmed. The opportunity to effect early mobilization of nearby joints is distinctly one of these.[2] Immediate metallic internal fixation of contaminated compound fractures has demonstrated its shortcomings. There has been learned, especially in such cases, the danger of loosening of plates and screws, of their inaccurate placement, of the absorption of bone at the fracture site resulting in apparent distraction and nonunion. External fixation splints, which depend for their supporting effect upon pins which penetrate the bone, have been tried and found wanting under battle conditions. Infection and persisting

sinuses about the pin-holes has been their chief disadvantage. Long immobilization in plaster casings, with its attendant stiffening of joints and atrophy of muscles, has been conclusively discarded in simple and compound fractures alike.

The need for prolonged support of the healed fractured femur by an unhinged weight-relieving caliper brace has been shown. The value of the walking encasement in minor fractures of the leg has been well demonstrated, as has the value of the "hanging cast" in certain fractures of the humerus. The extension splint with skeletal traction and functional position splinting in injuries to the bones of the hand has proven its merit.[7] Much, too, has been learned as to the unanticipated frequency of carpal bone fractures, especially of the navicular; of the necessity for their accurate diagnosis and appropriate and successful treatment by immobilization for periods as long as 16 weeks.

With the aid of penicillin much assurance has been gained in the successful prosecution of reparative procedures on bone. Its use in converting potentially infected compound fractures into clean, closed fractures has already been alluded to. Sequestrectomies, it has been learned, if completely done, may be safely followed by early wound closure or skin grafting, even in the presence of long-standing infection. Bone grafts and transplants may be accomplished at an earlier date than formerly with little fear of exacerbation of previously controlled infection. This emancipation from sepsis has largely removed the dread of long-continued or unhealing osteomyelitis.[8]

In compound injuries of the joints the plan of adequate débridement, cleansing, suture of the capule and open treatment of the remaining wound, accompanied by systemic penicillin or sulfa therapy, has well demonstrated its efficacy in preventing the dreaded complication of joint sepsis.

Bowel Surgery.—Surgery of the bowel has made progress as the result of war-time experience, both as regards the early management of injuries and of late reparative procedures. Suture and even resection of injured small bowel has been repeatedly performed with success, to which the bacteriostatic drugs have undoubtedly contributed. Exteriorizing colostomies have become consistently employed life-saving measures where the large intestine has been injured. Again and again there has been demonstrated the value of divided colostomies with complete exclusion of the fecal stream from distal damaged bowel. Complicated fecal fistulae, often with remote openings and associated osteomyelitis have been brought to healing when thus protected, and reparative procedures on rectum, perineum and bladder successfully accomplished. Chemotherapy has played its rôle well here, as also in the later intra-abdominal procedures so frequently needed to restore the integrity of the intestinal canal.[9]

With the assistance of these agents, and with appropriate proximal decompression, surgeons have learned no longer to fear for the result in resection and anastomosis of the large intestine. This attitude will no doubt hasten the acceptance of such procedures, already advocated by a number of surgeons, as applied to the surgery of cancer. Whether the internal use of sulfasuxidine

(succinylsulfathiazole) has contributed materially to the safety of these resections is questioned by some, but its repeated use for the purpose in war-time practice has demonstrated its value to the satisfaction of many surgeons employing it.

Thoracic Surgery.—Thoracic surgery, which in the years before the war had made such striking advances, has gained by fresh experience in the military hospitals. Early definitive surgery of chest wounds marks a substantial advance in this field which will surely carry over to civil practice. The early aspiration of hemothorax and the removal of organizing blood by thoracotomy, evacuation and, if necessary, decortication have diminished the incidence of pleural infection and have proven valuable measures for conserving lung function.[10] Still more frequently useful, no doubt, will be the method of early treatment of empyema by repeated aspiration and the instillation of penicillin solution. When coupled with the systemic administration of the drug many operations of thoracotomy for drainage will be avoided. The use of this drug, too, will appreciably reduce the hazards and complications of all pulmonary and intrathoracic surgical maneuvers.

Vascular Injuries.—The relative frequency of blood vessel injuries has led to a considerable experience in dealing with vascular occlusion states and with traumatic aneurysms and arteriovenous fistulae. The rôle of sympathetic innervation in influencing peripheral circulation and the values of sympathetic interruption in vasospasm are better understood. The importance of the distinction between progressive dissecting aneurysms with their threat of hemorrhage or pressure and the more stable arteriovenous aneurysms with their respective clinical and therapeutic implications, has been demonstrated and emphasized in experience.[20] The value of maximum safe delay to permit the establishment of collateral circulation has been repeatedly shown.

Neurosurgery.—Among the most striking lessons learned from war-time surgical experience have been in the field of neurosurgery. Here, again, the value of these experiences to civil practice will be particularly in the field of trauma. The gain in this is both qualitative and quantitative. By force of necessity the competent neurosurgeons have been obliged to devise and perfect methods of coping with a great variety of nervous system injuries and their sequelae. Moreover, great numbers of surgeons, previously inexperienced in this field, have become familiar with traumatic situations and have been obliged to treat them while learning by precept, example and experience. There can be little doubt that the brain and nerve injuries of civil life will be better treated by better equipped surgeons in consequence.

In the management of compound head injuries the principles of careful investigation, thorough removal of bone fragments and foreign bodies, suction débridement, accurate hemostasis and preservation and repair of the dura have been thoroughly inculcated.[11] So, also, has the principle of early and accurate peripheral nerve repair or, at least, identification. The need for functional, protective support of denervated muscles has come to be well

recognized, whether accomplished by mechanical splinting or by surgical means. In the field of reparative neural surgery advances have been most significant in the evaluation of brain-compromising scars and their removal, in the repair and replacement of injured dura, often with the aid of fibrin film, and in the closure of skull defects with metallic plates. The technics of neurolysis and suture repair of peripheral nerves have undergone great improvement, to which the employment of tantalum wire sutures and fibrin glue have added much. The use of fibrin foam as an hemostatic agent greatly facilitates the work of the neurosurgeon.[12] Much, too, has been learned concerning the employment of nerve grafts, although the results of grafts of major nerves have not been encouraging to date. This fact has led to the development of considerable skill and ingenuity in securing end-to-end union of severed and shortened nerves by neurolysis, transplantation, posture and even bone shortening.[14] In all neurosurgical work the perfecting of the investigative procedures of encephalography and neuromyelography have played a considerable part.

Striking advance has been made in the treatment of those unfortunate patients who have suffered paraplegias resulting from spinal cord injury. General supportive measures have restored their nutrition, cystostomies have been closed and automatic bladders established. Bedsores have been cleaned and healed, often by suture. Directed exercise and training have restored the strength of unparalyzed muscles. Braces have been fitted and patients trained in their use to the extent that many of them have actually become ambulant in spite of what has usually, in the past, proved a completely incapacitating and frequently fatal disability.

Plastic Surgery.—Plastic surgery, long the stepchild of various specialties, has come into its own by virtue of the stimulus of war-time needs. While standard fundamental procedures of repair have not been changed, new applications and adaptations have needed to be devised, accepted and taught. The disfigurements and disabilities produced by war's injuries have stimulated and taxed the ingenuity of plastic surgeons to the full. That they have profited by this experience and that many additional competent surgeons have received valuable instruction in this important field cannot be doubted. Three developments, in particular, have added to the effectiveness of this type of surgery. They are the perfecting of methods for the utilization of stored or refrigerated skin grafts[13]; the adaptation of plastic substances, notably acrylic, for the replacement of structural defects about the face; and the use of tantalum wire in place of fascia to replace the action of denervated muscle groups.

Elective Surgery.—Experience in time of war with certain nonemergency surgical procedures has given an invaluable opportunity to review their results in large numbers of cases. In this group are the operations for protruding intervertebral disk or herniated nucleus pulposus. Much controversy has developed concerning this procedure, as to its indications, technic and results. From it all has come a generally acceptable point of view, less

enthusiastic than that of some of its more radical proponents, less conservative than that of some of its detractors. This point of view may be expressed as requiring demonstrable neurologic and roentgenologic demonstration of the lesion as indications for operation. Army experience has led to the conclusion that in such cases surgical relief of the condition may be expected to result in improvement in about 60 per cent of the cases, but only after the passage of time and with protection of the patient from lifting, straining and excessive back motion. The need for a considerable postoperative period of restricted activity has been quite clearly indicated.

Traumatic internal derangements of the knee have been encountered quite frequently in military practice. From them one lesson, in particular, has been given emphasis. That is the necessity of preserving the stability of the knee joint by meticulous care in protecting the capular ligaments (especially the internal lateral ligament) from injury. The value of preoperative conditioning by exercise of the muscles about the knee joint, especially the quadriceps femoris, has been demonstrated in promoting earlier recovery and restoration of function after surgery.

About the matter of pilonidal disease there has also been a crystallizing of opinion. It is apparent that a policy of operative removal of every such defect discovered will not pay dividends in conservation of time and comfort. The self-regulating mechanism of civil life which deters the victim of such a defect from consulting his surgeon unless sufficient discharge or the development of inflammation renders him so uncomfortable as to seek relief, can be counted upon to control any excessive performance of the operation for removal of the lesion. In such cases as may require removal because of repeated inflammation, opinion is still somewhat divided between open and closed procedures. In appropriate cases, especially with the help of penicillin, the closed method has undoubtedly produced many excellent early results. Elaborate flap and plastic closures have gone out of fashion.

Surgical Complications.—Certain surgical complications have been successfully combated by measures which, already known, have shown their value in the intensive experience of war-time practice. Thus, most surgeons have become convinced of the desirability of intubation drainage in dealing with postoperative pulmonary atelectasis. The early postoperative institution of bed exercises and deep breathing have shown merit as preventive measures. The general use of spinal as opposed to inhalation anesthesia seems to have shown little effect on the incidence of this complication. In pneumonitis penicillin has time and again proven its value.

Similarly, a better understanding of peripheral phlebothrombosis has been disseminated. Most Army surgeons are now trained closely to observe their wounded and postoperative patients for the early detection of the swelling, calf tenderness and pain on plantar foot flexion that betoken the onset of clotting in the deep leg veins, and, finding them, to forestall the deadly threat of embolism by early femoral vein ligation.

Another contribution is the development of the treatment of bedsores.

Recognition and correction of their contributing causes—pressure, starvation, hypoproteinemia, soiling, dehydration, anemia and avitaminosis—sets the stage for their definitive treatment. Cleansing, protective dressings and the rapid disposal of slough, sometimes with the help of urea crystals or granulated sugar or both, prepares the sore for closure by wire sutures after undermining and approximation of the skin edges. Systemically administered penicillin, before and after operation, contributes to the complete and clean healing that occurs in nearly 65 per cent of cases thus treated.[14]

Materials.—The exigencies of war-time surgery have stimulated the development and popularized the use of a number of materials which will contribute much to the welfare of patients in civilian peace-time practice. The brilliant researches of the laboratory in the fractionation of blood proteins have given us serum albumin, so valuable in shock and protein deprivation. They have provided fibrin foam so effective as a local hemostatic agent, particularly in neurosurgery and in dealing with vascular visceral structures, especially the liver; fibrin film, useful as a tissue protective and replacement agent, especially for the scarred or injured dura; and thrombin-fibrinogen glue used in peripheral nerve suturing and skin grafting.[15] Tantalum has been popularized as a nonreactive, electrically inert, yet strong and workable material as wire for sutures, plates for bone defects, and foil for protection of delicate tissues.[16] The general use of nonabsorbable sutures of wire, cotton and silk has won many converts and has set the surgical style for at least a generation to come. Acrylic (methyl methacrylate resin) has been developed as a strong, light, nonreactive and nonsoluble material for the making of dentures, eyes and prosthetic appliances useful in plastic surgery.

Supportive Measures.—In dealing with large numbers of seriously ill and injured patients in war-time there has been developed a fresh appreciation of the importance of certain general supportive measures to be employed not only as a postoperative procedure and in protracted illness, but as means of prevention of deterioration and as means of preoperative preparation. In this direction, in addition to a recognition of the need for nutritive factors, fluid and electrolytes, new emphasis has been placed on the necessity of forestalling and controlling vitamin deficiency states. The effects of the avitaminoses on nutrition, wound healing and resistance to infection have been made increasingly clear. Correspondingly, there is a more general appreciation of the effects of fatigue, sepsis and malnutrition in producing the vicious circle of avitaminosis. The values of the B-complex of C and D have been repeatedly demonstrated in preventing and correcting such situations, and of K in the prophylaxis and control of hemorrhagic states.

The importance of maintaining nitrogen balance is now more fully appreciated and the means for correcting its lack are at hand in plasma, serum, albumin and the amino-acid preparations. The supportive and corrective values of repeated blood transfusions have been reaffirmed, and additional safeguards for this type of therapy have been developed especially in the recognition of the importance of the *Rh* factor.[17] The red cell by-product

of the plasma production program has found increasing use, employed in suspension in the restoration of hemoglobin deficiencies[18] and, in the hands of some surgeons, seems to have been beneficially utilized locally in open-wound dressings.

Reconditioning.—By no means the least valuable contribution of war surgery is a new concept of the value of a rehabilitation program. Faced by the need of conserving manpower, the Army rapidly began a plan of reconditioning the hospitalized soldier the object of which was to restore him to health and vigor and return him to military duty with the least waste of time. This program's activities begin with the patient still in bed and as soon after operation as his condition will permit. General bed exercises are given, and special exercises prescribed for restoration of the affected part. Thus, the patient with a leg lesion receives not only exercises of arms, trunk, neck and back, but is drilled specifically in the quadriceps setting exercises which serve to prevent weakness and restore muscle and joint function even though he be confined to bed. In certain types of cases, notably internal derangements of the knee, these special exercises have been given preoperatively, as a prophylactic preparation, with great benefit. It is the universal impression, as the result of experience, that such bed exercising, both general and special, have hastened recovery, shortened bed rest and hospitalization and have tended greatly to diminish the incidence of pulmonary and thrombotic vascular complications.

The program of exercise and increasing directed physical activity has been pursued after the patient has left his bed, through remedial and corrective calisthenics and gymnastics, occupational therapy adapted to his particular needs, and recreational activities. This program has been worked out with care and diligently pursued to the point where the patient, fully restored, has been made ready to resume his duties, after minimum hospitalization. The benefits of this war-developed practice have been so fully demonstrated as to affect profoundly our attitude toward the management of patients in peace-time practice, and throw into sharp contrast the general neglect of such measures in the past.[19]

This program of reconditioning has not involved the patient's physical status alone. Recreation, instruction, amusement, the development of new skills and hobbies, the stimulation of new interests and a new outlook have played a large part in it. The importance of this phase of rehabilitation, particularly as it applies to the permanently handicapped, can hardly be overemphasized. The feeble and tentative efforts hitherto made in this direction for our civilian patients will undoubtedly be greatly increased and strengthened in the future as the result of war experience.

In presenting this general summary and review no attempt has been made to cover the many technical details and refinements of procedure that have developed in various commands and hospitals as the result of war experience. It is not intended to convey the impression that the advances achieved have been due solely to war nor to the activities of Army surgeons alone. Many

had already been instituted and perfected in civilian hospitals and laboratories, and had even been quite widely recognized. It is fair to say, however, that the experiences of war have led to a more rapid proving and evaluation of their merits, the acceptance of some and the rejection of others. Certainly the school of war-time surgery has served to disseminate rapidly throughout the body of the profession a considerable fund of information which, in the long run, cannot but work to the profit of peace-time practice.

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